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ON THE
PHYSIOLOGY OF THE SEMICIRCULAR CANALS AND THEIR RELATION TO SEASICKNESS

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PHYSIOLOGY OF THE SEMICIRCULAR CANALS AND THEIR RELATION TO SEASICKNESS

BY

JOSEPH BYRNE, A.M., M.D., LL.B.

NEW YORK
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TO

DAVID FERRIER

A PIONEER IN THE PHYSIOLOGY OF THE NERVOUS
SYSTEM, WHOSE CAREFUL EXPERIMENTS, ACCURATE
OBSERVATIONS, AND SOUND DEDUCTIONS RENDER
HIS WORK A GUIDE AND INSPIRATION FOR STUDENTS
OF GENERATIONS TO COME

PREFACE

Six years ago the author undertook to write an article on the etiology of seasickness. Not satisfied with a mere expression of views without appeal to experimental fact, and believing that the semicircular canals were in some way involved in the causation of the malady, he undertook of his own suggestion and forethought, a series of experiments using rotations, aural irrigations, stimulation of the retina by strong light, galvanism applied to the mastoid areas, etc., to determine whether by such means phenomena resembling those of seasickness could be experimentally reproduced. The results of his earliest experiments fulfilled his expectations to such an extent, that he proceeded to a thorough study of the semicircular canals, as affected by rotations, aural irrigations and galvanism.

The phenomena of nystagmus and the displacements of the head that occur in rotations, aural irrigations, and galvanism applied to the mastoid areas, were observed by him independently and the mechanisms involved in their production studied and worked out before he had any information of the work done by Bárány, Neumann and others. He makes therefore, no pretensions for this part of his work on the score of priority in time, but submits it to the profession in the hope that the work will speak for itself.

Armed with the knowledge and experience gained from a thorough study of the sickness produced by rotations, aural irrigations, etc., he next proceeded to the experimental study of seasickness, the results of which are detailed in Part III.

Although numerous volumes were consulted the author owes special acknowledgment to the works of Ferrier, Sherrington, Piersol, Risien-Russell, and Ewald. The description of the labyrinth and of the eighth nerve is taken mainly from Piersol's Human Anatomy.

The author wishes to thank Dr. Bailey and Messrs. William Wood & Co. for permission to reproduce three figures from Dr. Bailey's work on Histology. He also wishes to thank his friend Mr. Hugh J. Smith for the patience and fortitude with which he submitted to the various tests, as well as for the intelligent assistance given in describing the subjective phenomena.

The author has always been an acute sufferer from seasickness, and the time and labour devoted to these investigations, which were conducted in private, he considers well spent if his efforts tend to a better understanding of this distressing malady, with consequent alleviation of unspeakable human misery.

J. BYRNE.

29 West 61st Street, New York City,
January, 1912.

CONTENTS

PART I

GENERAL ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS

CHAPTER PAGE
I. ANATOMY OF THE LABYRINTH 3
II. THE EIGHTH NERVE 12
III. THE REMAINING CRANIAL NERVES 22
IV. BLOOD-SUPPLY OF THE LABYRINTH AND OF THE CRANIAL NUCLEI 37
V. SYNOPSIS OF ANATOMICAL CONNECTIONS . . . 40
VI. THE SYMPATHETIC OR AUTONOMIC NERVOUS SYSTEM 98
VII. THE PATHS INVOLVED IN PUPILLARY MOVEMENTS 105
VIII. FURTHER ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS 114

PART II

PHYSIOLOGY OF THE SEMICIRCULAR CANALS

IX. PHYSIOLOGY OF THE SEMICIRCULAR CANALS FROM THE STANDPOINT OF ANIMAL EXPERIMENTATION 125
X. THE EFFECTS OF PASSIVE ROTATION . . . 130

CHAPTER PAGE
XI. THE EFFECTS OF ROTATION UPON THE DIGESTIVE APPARATUS 164
XII. THE EFFECT OF DRUGS AND OTHER MEASURES UPON DERANGEMENTS OF THE ALIMENTARY SYSTEM CAUSED BY ROTATION SICKNESS . . 183
XIII. THE EFFECT OF ROTATION UPON EQUILIBRIUM 192
XIV. THE EFFECTS OF ROTATION UPON THE EYES . 198
XV. THE GENERAL EFFECTS OF AURAL IRRIGATIONS 207
XVI. THE EFFECTS OF AURAL IRRIGATIONS UPON THE DIGESTIVE APPARATUS 233
XVII. THE EFFECTS OF AURAL IRRIGATIONS UPON THE EYES 250
XVIII. THE EFFECTS OF THE GALVANIC CURRENT UPON THE SEMICIRCULAR CANALS 268
XIX. HOW ROTATIONS, AURAL IRRIGATIONS AND GALVANISM AFFECT THE LABYRINTHINE RECEPTORS AND THE RELATED EFFECTORS . . . 273
XX. MECHANISM OF THE NYSTAGMUS OF ROTATIONS, AURAL IRRIGATIONS AND GALVANISM . . . 288
XXI. RELATIONS OF THE SEMICIRCULAR CANALS TO THE OCULO-MOTOR NUCLEI AND THEIR BEARING UPON THE RESULTS OF CERTAIN EXPERIMENTS 306
XXII. ON OCULAR MOVEMENTS AND NYSTAGMUS . . 315
XXIII. ON THE OCCURRENCE OF NYSTAGMUS . . . 331

PART III

SEASICKNESS

XXIV. STUDIES IN SEASICKNESS 339
XXV. FURTHER STUDIES IN SEASICKNESS DURING A TRANSATLANTIC TRIP 345
XXVI. STUDIES IN SEASICKNESS (CONTINUED) . . . 357

CHAPTER PAGE

XXVII. Studies in Seasickness (Continued) . . . 379

XXVIII. Studies in Seasickness (Continued) . . . 384

XXIX. General Conclusions from Studies in Seasickness. Protocols 417

XXX. History and Literature of Seasickness . 478

XXXI. Etiology of Seasickness 486

XXXII. Etiology of Seasickness (Continued) . . 496

XXXIII. On the Occurrence of Seasickness . . . 508

XXXIV. The Effects of Seasickness and Their Relation to Diseased Conditions 512

XXXV. How Recovery from Seasickness Takes Place. Outlines of Treatment . . . 518

BIBLIOGRAPHY 527

INDEX 537

PART I

GENERAL ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS

CHAPTER I

ANATOMY OF THE LABYRINTH

The long axis of the internal ear measures about 20 mm, and corresponds with that of the petrous bone. The cavity of the bony labyrinth is divided into an anterior portion, the cochlea, a middle portion, the vestibule, and a posterior portion, the semicircular canals. The vestibule is an irregularly elliptical cavity measuring about 5 mm from before backward, the same from above downward, and about 3—4 mm from without inward. The outer wall constitutes that part of the inner wall of the tympanic cavity in which the oval window is situated. The medial or inner wall is directed toward the bottom of the internal auditory canal, and has two depressions separated by a ridge—the crista vestibuli, the upper pointed end of which forms the pyramidalis vestibuli. The anterior, the smaller of these depressions, is the spherical recess and lodges the saccule. In the lower part of this fossa a number of perforations (about a dozen) mark the macula cribosa media, through which pass, from the bottom of the internal auditory canal, the branches of the vestibular nerve to the saccule. The posterior, larger depression, is the elliptical recess, which lodges the utricle. Behind the lower part of the spherical recess the crista vestibuli divides into two limbs, between which is the recessus cochlearis, which lodges the beginning of the ductus cochlearis, and is pierced by a number of small openings for passage of the nerve filaments to this duct. The openings in the crista vestibuli and the elliptical recess collectively form the macula cribrosa superior, and transmit branches of the vestibular nerve to the utricle and to the ampullæ of the superior and horizontal semicircular canals. Below and behind the recessus ellipticus lies a groove, the fossa sulciformis, which deepens pos-

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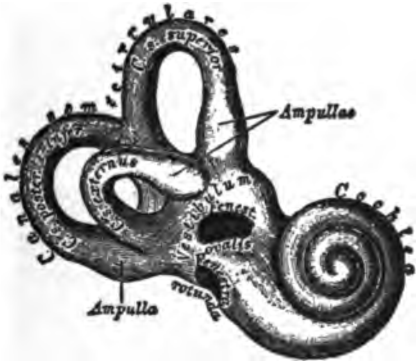
The semicircular canals are three small bony tubes, of a shape indicated by their name. They constitute the posterior division of the bony labyrinth. The direction of each canal corresponds roughly with one of the fundamental planes of the body, viz., the superior canal corresponds with the coronal or frontal, the posterior with the sagittal, and the horizontal with the transverse. The plane of their direction is such that each canal is at right angles to the direction of each of its fellows. At one end of each canal there is a dilated portion—the ampulla. The superior canal lies farthest front, and in nearly a vertical plane at right angles to the long axis of the petrous bone. The plane of the posterior, which is the longest of the canals, is approximately parallel to the long axis of the petrous bone. The external portion of the horizontal semicircular canal forms a prominence in the inner wall of the middle ear, behind the facial canal, while the upper part of the superior semicircular canal produces a conspicuous elevation—the eminentia arcuata, seen on the superior surface of the petrous bone. The canals open into the posterior part of the vestibule by five openings, the undilated ends of the superior and posterior canals uniting to form a common trunk, the crus commune. The horizontal canal alone has two distinct openings into the vestibule. Its ampulla is at its outer end, and lies at the upper part of the vestibule, above the oval window, from which it is separated by a groove corresponding with the facial canal.

The ampullary end of the posterior canal lies on the floor of the vestibule, near the opening of the undilated end of the horizontal canal and that of the canalis communis. The ampulla of the superior canal lies in the vicinity of the ampulla of the horizontal canal, but is situated somewhat mesial to it. This is an important relation in view of the fact that stimuli by means of irrigations in the external auditory canal, or by means of galvanism over the mastoid area, usually affect the ampulla of the horizontal canal only; but if thermic irritation be long continued, or if there be structural changes due to long-continued disease of the middle ear, phenomena indicating irritation of the ampulla of the superior canal may appear simultaneously with those from irritation of the ampulla of the horizontal canal, or may even be present to the exclusion of the latter. In the wall of the ampulla of the posterior canal a number of small openings constituting the macula cribrosa inferior provide for entrance of the special branch of the vestibular nerve, destined for this canal. Sappey[1] states that the superior canal bends somewhat upon itself, so that the anterior (external) half inclines a little inward (*en dedans*), and the posterior (internal) half inclines a little outward (*en dehors*). Poirier and Charpy[2] follow Sappey's description, which is undoubtedly accurate.



The Bony Labyrinth. ×3. (Heitzmann.)

(*From "Histology," by Dr. Bailey. Wm. Wood & Co., Publishers*)

The membranous labyrinth lies within the bony labyrinth, and resembles it in general form. This agreement is least marked in the vestibule, since here the bony capsule is occupied by two compartments of the membranous sac—the utricle and saccule. The membranous labyrinth comprises:

1. The utricle and saccule, which, with the ductus endolymphaticus, lie within the vestibule.
2. The three membranous, semicircular canals, which lie within the bony canals.
3. The membranous cochlea enclosed within the bony cochlea.

The membranous labyrinth is attached especially at certain places by connective tissue to the inner wall of the bony capsule. The space between the membranous and bony labyrinths—largest in the scala tympani and scala vestibuli of the cochlea and in the vestibule—constitutes the spatium perilymphaticum, and contains a modified lymphatic fluid, the perilymph. The fluid which fills the interior of the membranous labyrinth is called the endolymph, and can pass from one part of the labyrinth to another, although the saccule and utricle are only indirectly connected through a narrow channel—the ductus endolymphaticus. The utricle occupies the recessus ellipticus in the upper and back part of the vestibule. Larger than the saccule, it communicates with the three membranous semicircular canals. It is attached to the upper and inner walls of the vestibule by connective tissue. It extends from the roof of the vestibule backward and downward to the opening of the posterior ampulla (5.5 to 6 mm).

The utricle has three subdivisions, the uppermost a blind sac (3 to 3.5 mm in length and breadth), called the recessus utriculi, whilst the two lower divisions form the utriculus proprius, which measures 3 mm by 1.5 to 2 mm. The lower part of the utricle proper is prolonged into the tube-shaped sinus posterior, which connects the ampulla of posterior canal with the utricle.

The openings of the semicircular canals into the utricle are as follows: (a) Into the recessus utriculi: the ampullæ of the superior and horizontal canals. (b) Into the utriculus proprius:

1. The sinus superior, which lies within the crus commune, and receives in turn the non-ampullated ends of the superior and posterior canals.

2. The non-ampullated end of the horizontal canal.

3. The ampulla of the posterior canal through the sinus posterior.

On the antero-lateral wall of the recessus utriculi is placed the macula acustica of the utricle, whilst from its antero-mesial wall springs the canalis utriculo-saccularis, the small canal from the utricle that joins a still smaller passage from the saccule to form the ductus endolymphaticus.

The saccule is an irregularly oval compartment, about 3 by 2 mm, occupying the recessus sphæricus in the lower and anterior part of the vestibule, to which it is attached by connective tissue. It is somewhat flattened laterally, and at its lower end gradually narrows into a passage—the canalis reuniens, which connects the saccule with the ductus cochlearis. Its upper end bulges backward, forming the sinus utricularis, the wall of which comes in contact with that of the utricle. The small canal that helps to form the ductus endolymphaticus springs from the posterior wall of the saccule. The ductus endolymphaticus passes through the aquæductus vestibuli to end in a blind dilated extremity, the saccus endolymphaticus, lying between the layers of the dura mater below the opening of the aqueduct. Through openings in the recessus sphæricus branches of the vestibular nerve enter and pass to the macula acustica sacculi on the anterior wall of the saccule. The canalis reuniens is the very small tube passing from the lower part of the saccule into the upper wall of the cochlear duct, near the cæcum, as its blind vestibular end is called.

The membranous semicircular canals (ductus semicirculares) occupy about one-third of the diameter of the osseous canals, and correspond with them in number, name, and form. They are closely united along their convex margins with the bony tubes, whilst the opposite concave margins lie free in the perilymphatic space, being attached only by irregular vascular connective-tissue bundles (ligamenta labyrinthi canaliculorum), which stretch across this space. Each of the membranous tubes has an ampulla, which is relatively much larger than the osseous ampulla, being three times the size of the rest of the tube. The part of the ampulla corresponding to the convexity of the semicircular canals is grooved on the outer surface at the entrance of the ampullary nerves. On the corresponding inner surface is a projection—the septum transversum—which practically divides this space into two parts, and is surmounted by the crista acustica, which contains the ampullary endings of the vestibular

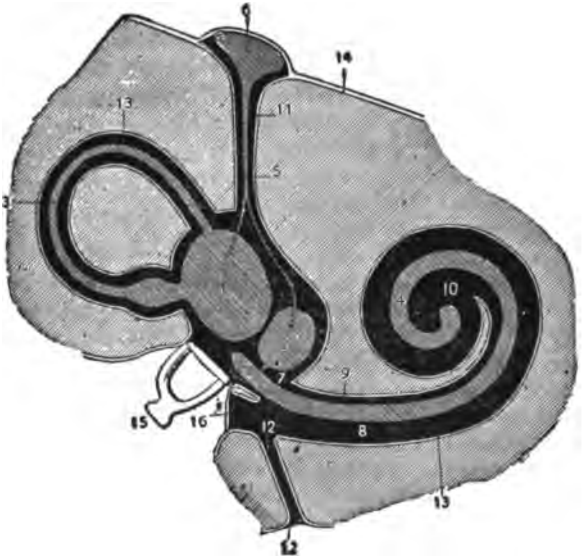


Diagram of the Perilymphatic and Endolymphatic Spaces of the Inner Ear. (Testut.) Endolymphatic spaces in grey; perilymphatic spaces in black. 1, Utricle; 2, saccule; 3, semicircular canals; 4, cochlear canal; 5, endolymphatic duct; 6, subdural endolymphatic sac; 7, canalis reuniens; 8, scala tympani; 9, scala vestibuli; 10, their union at the helicotrema; 11, aqueduct of the vestibule; 12, aqueduct of the cochlea; 13, periosteum; 14, dura mater; 15, stapes in fenestra ovalis; 16, fenestra rotunda and secondary tympanic membrane.

(From "Histology," by Dr. Bailey. Wm. Wood & Co., Publishers)

nerves. The crescent-shaped thickening beyond each end of the crista is called the planum semilunatum.

Structure of the Utricle, Saccule, and Semicircular Canals.

The vestibule and the bony semicircular canals are lined by a very thin periosteum, composed of a feltwork of resistant fibrous tissue containing pigmented connective-tissue cells. Endothelium everywhere lines the perilymphatic space between the membranous and osseous canals, covering the free inner surface of the periosteum, the fibrous trabeculæ, and the outer or perilymphatic surface of this part of the membranous labyrinth. The walls of the utricle, saccule, and membranous semicircular canals are made up of (a) an outer fibrous connective-tissue lamella, and (b) an inner epithelial lining, the latter consisting throughout the greater part of its extent of a single layer of thin, flattened, polyhedral cells. Beneath the epithelium, in the region of the maculæ and cristæ, is (c) a thin, almost homogeneous, hyaline membrane with few cells. This middle layer presents, in places, on its inner surface small papillary elevations covered by epithelium. On the concave side of the semicircular canals is a strip—the raphé—of thickened epithelium, in which the cells become low, cylindrical in type. In the plana semilunata the cells are cylindrical in type. Over the regions receiving the nerve fibres—the maculæ acusticæ and the cristæ acusticæ—the epithelium undergoes a marked alteration, changing from the indifferent covering cells into the highly specialized neuro-epithelium.

The maculæ acusticæ are about 3 mm long by 2 mm broad, the macula of the saccule being a little narrower (1.5 to 1.6 mm) than that of the utricle (2 mm). At the margin of these areas the cells are at first cuboidal, next low columnar, and then they abruptly increase in length until they measure from 0.30 to 0.35 mm, in contrast with their usual height of from .003 to .004 mm. The acoustic area includes two kinds of elements—the sustentacular or fibre cells and the hair cells. The sustentacular cells are long, rather narrow, irregularly cylindrical elements, and extend the entire thickness of the epithelial layer, resting upon a well-developed basement membrane by their expanded or divided basal processes. At a variable distance from the base they present a swelling enclosing an oval nucleus, and terminate at the surface in a cuticular zone. The cylindrical hair cells are broader but shorter than the sustentacular cells, and reach from the free surface only as far as the middle of the epithelial layer, where each cell terminates usually in a rounded or somewhat swollen end, containing a spherical nucleus. The end next to the free surface exhibits a differentiation into a cuticular zone, similar to that covering the inner ends of the sustentacular elements.

From the free border of each hair cell a stiff, robust hair, .020 to .025 mm long, projects into the endolymph. This, however, is resolvable into a number of agglutinated, finer hairs or rods. The free surface of the neuro-epithelium within the saccule and utricle is covered by a remarkable structure, the so-called otolithic membrane. This consists of a gelatinous membrane, in which are imbedded numberless small, crystalline bodies, the otoliths. Between it and the cuticular zone is a space, about .020 mm in width and filled with endolymph, through which the hair cells pass to the otolithic membrane. The otoliths are minute crystals, usually hexagonal in form, with slightly rounded angles, and from .009 to .011 mm in length. They are composed of calcium carbonate, with an organic basis. On reaching the macula the nerve fibres form a subepithelial plexus, from which fine bundles of fibres pass toward the free surface. The fibres usually lose their medullary substance in passing through the basement membrane, and enter the epithelium as naked axis-cylinders. Passing between the subcuticular cells to about the middle of the epithelium, they break up into fine fibrillæ, which embrace the deeper ends of the hair cells, and give off fine threads that pass as free axis cylinders between the cells to higher levels.

The crista acustica and planum semilunatum are covered with neuro-epithelium, similar to that of the maculæ. The hairs of the hair cells, however, are longer and converge to, and are imbedded within, a peculiar domelike structure known as the cupola, regarded by some anatomists as an artefact formed by coagulation of the fluid in which the hair cells are bathed. Otoliths probably do not exist in the cristæ acusticæ.

In some lower animals, besides the maculæ of the utricle and saccule, there is also a third structure similar to these—the lagena—which likewise contains otoliths. In certain fishes the otoliths are stones of 1 cm in size. The plane of the macula utriculi corresponds roughly with that of the external semicircular canal. In the upright position it is inclined backward at an angle of 45°. According to Retzius[3] the long axis of the macula utriculi in men is directed from in front upward and inward and (the short axis) backward, downward, and outward, the general plane being about horizontal. The long axis of the saccule is almost vertical, and is directed from without upward and inward, the short axis being directed from before backward. The macula is situated upon its mesial surface (Retzius). The plane of the macula sacculi runs from above and behind forward and downward in an antero-posterior direction, at about an angle of 45°. The lagena of lower animals permits vertical displacement of its otolith, while the maculæ of the utricle and saccule are roughly horizontal, and at right angles to each other (Piersol[4]). The hairs of the maculæ of the utricle and saccule are stiffer and shorter than those of the cristæ of the ampullæ, and are held together by a stiffer mass of otoconium, which, however, is not gelatinous, but of otolithic composition. Verworn[5] proposed the name staloliths for the otoliths of the utricle and saccule, and called the analogous organs in certain lower forms statocysts, thus assuming that the function of these organs is exclusively static, as distinguished from the dynamic function of the semicircular canals. Clarke,[6] after a comparative study of the otolithic and related structures, concludes that whilst the otolithic structures are mainly statical in function, they are not exclusively so, and may function dynamically as well. With the exception of the maculæ of the utricle and saccule no part of the vestibular wall is supplied with nerve terminals of the eighth nerve.

CHAPTER II

THE EIGHTH NERVE

The eighth nerve consists of two portions—the cochlear, or true nerve of hearing, and the vestibular, which is concerned with equilibration. Traced from the brain toward the ear, the eighth nerve arises at its superficial origin by two roots: a mesial—radix vestibularis—and a lateral—radix cochlearis—which embrace the inferior cerebellar peduncle, the mesial root passing to the inner, and the lateral root to the outer side of the peduncle. The nerve thus formed by the union of these two roots leaves the surface of the brain-stem at the posterior border of the pons, where it is adherent to the middle cerebellar peduncle. To its inner side, and closely associated with it, are the motor and sensory roots of the facial nerve, which lie within a groove on the mesial surface of the eighth, and with it enter and traverse the internal auditory canal. Within the latter the eighth nerve separates into two divisions, of which the superior and larger is the vestibular nerve (n. vestibuli), and the inferior and smaller the cochlear (n. cochleæ). Although in a general way these divisions continue the corresponding roots, this agreement as to the source of their fibres is not complete, since strands of the vestibular fibres are incorporated with the cochlear nerve. On reaching the bottom of the internal auditory canal, the facial nerve enters the facial canal, whilst the fibres of the eighth nerve disappear through apertures in the lamina cribrosa, to reach the several parts of the membranous labyrinth. In the internal auditory canal the vestibular and facial trunks are connected (fila anastomica) by a branch, which passes from the pars intermedia to the vestibular nerve, and by one from the latter to the geniculate ganglion. These apparent communications between the seventh and eighth nerves are, in fact, only aberrant strands of facial fibres that return to the seventh after temporary association with the eighth.

The vestibular nerve divides into three terminal branches, which pass through the apertures in the cribriform plate, above the falciform crest, and supply the utricle and the superior and external semicircular canals. Not all the fibres of the vestibular root, however, are included in these branches, since of the three branches given off by the vestibular nerve, two, viz., those of the saccule and posterior semicircular canal, are incorporated with the cochlear fibres, and seemingly are derived from the cochlear nerve. The remaining branch of the cochlear nerve contains the cochlear fibres proper, which traverse the numerous foramina of the tractus spiralis foraminosus and the central canal of the modiolus, to supply the organ of Corti.

The fibres of the eighth nerve are afferent. Hence they are processes (axones) of nerve cells, situated somewhere along the course of the nerve. The real origin of the nerve fibres, therefore, is to be sought in the ganglia, occurring in the divisions of the nerve.

The true cochlear fibres arise within the cochlea as axones of the cells of the spiral ganglion, or ganglion of Corti (g. spirale). This structure consists of a series of bipolar neurones, which occupies the spiral canal in the base of the lamina spiralis. The dendritic processes of these cells begin as fine fibrils, which lie in close relation with the neuro-epithelial cells, comprising the inner and outer hair cells of the organ of Corti. Leaving the hair cells as non-medullated fibres, they traverse the foramina nervosa of the labium tympanicum, at which point they become medullated when they interlace to form a flat feltwork that lies between the layers of the lamina spiralis, and soon assemble to form bundles which pass to the cells of the ganglion spirale, each fibre probably joining its individual cell. Leaving the ganglion, the axones of its cells enter the bony canals within the modiolus, from which they emerge as the tractus spiralis foraminosus, and are collected into a single bundle—the cochlear nerve proper. This latter, however, soon receives two accessions, one of which consists of fibres from the saccule, the other from the posterior semicircular canal. These accessions are in reality parts of the vestibular nerve and, beyond their temporary companionship have nothing to do with the cochlear root.

On reaching the medulla the cochlear fibres come into relation with their nucleus of reception, which includes two superficial aggregations of nerve cells that collectively constitute the acoustic nucleus. The latter consists of two parts, of which one—the ventral cochlear nucleus, also called the accessory acoustic nucleus—lies ventral to the inferior cerebellar peduncle; and the other—the lateral cochlear nucleus, or tuberculum acusticum, rests upon the dorso-lateral surface of the peduncle, and occupies the extreme outer part of the triangular acoustic area, seen in the lateral angle of the floor of the fourth ventricle. The greater number of cochlear fibres end in arborizations around the stellate cells of the ventral ganglion, whilst others terminate in relation with the more elongated fusiform cells of the lateral nucleus. From the neurones of these subdivisions of the reception nucleus, the auditory pathway is continued as two chief tracts—the axones of the cells of the ventral nucleus passing for the most part ventral to the restiform body and the special root of the trigeminus to form the corpus trapezoides, whilst those from the lateral nucleus sweep around the outer surface of the restiform body, and then medially beneath the ependyma of the floor of the fourth ventricle, where they show with varying degrees of distinctness as the striæ acusticæ. The corpus trapezoides—the conspicuous transverse tract that separates the tegmental from the ventral region of the pons in its superior part—is formed chiefly by the axones of the cells within the central cochlear nucleus, supplemented by a limited number of fibres that spring from the lateral nucleus. In addition it contains axones from the large cells found within the trapezoid body on each side of the middle line that constitute the nucleus trapezoideus. In close relation with the dorsal surface of the corpus trapezoides, within the superior olive and on either side of the median raphé, lies the superior olivary nucleus (nuc. olivarius superior), a collection of nerve cells around which many of the cochlear fibres, chiefly from the opposite but also from the same side, end and from which the tract of the lateral fillet principally takes origin. Not all the fibres arising from the superior olivary nucleus, however, enter the lateral fillet. A considerable number leave the dorsal surface of the nucleus, and as its peduncle pass to the abducent nucleus (v. Bechterew considers these fibres as cerebellofugal paths to the abducens nucleus), and by way of the posterior longitudinal fasciculus to the nuclei of the other ocular nerves. In this manner it is asserted the reflex paths are established, by which the motor nerves, including probably the facial, are brought under the influence of auditory impulses.

There are reasons, however, for rejecting this simple, almost direct relation between the eighth nerve and the nuclei of the eye muscles. Movements of the eyes in response to a loud sound are not simple reflex movements, but complex, coordinated movements, associated usually with other movements, e.g., turning of the head and eyes to the source of the sound, accompanied by a general movement of withdrawal of the head and upper part of the body. Such complex purposive movements could hardly take place through direct paths from the auditory to the ocular and other nuclei without the intervention of some higher harmonising mechanism. Moreover, in Ferrier's[8] experiments, stimulation of the auditory area in the temporal lobe caused the animal to turn the head and eyes in the direction of the ear of the opposite side. This interesting experiment shows that the main relation between the auditory nerve proper and the cerebral cortex is a crossed one. It also shows that reflex, coordinated turning of the head and eyes in response to auditory stimuli may take place along other paths than the direct one mentioned; that is, in a downward course, possibly through the medium of the cerebellum.

Within the tract of the fillet, a short distance beyond the superior olive, is encountered a group of nerve cells, the nucleus of the lateral fillet (nucleus lemnisci lateralis). Whilst numerous additions to the fillet are received from these cells, their relation to the cochlear fibres is uncertain. The lateral fillet will be more fully described later on. It is sufficient to note here that in so far as the auditory (cochlear) fibres are concerned, the tract terminates chiefly in the inferior colliculus of the quadrigemina and in the median geniculate body. In addition to its constituents through the corpus trapezoides the lateral fillet receives considerable accessions of cochlear fibres by way of the striæ acusticæ. These strands consist, for the most part, of the axones of the cells lying within the tuberculum acusticum; but to a limited extent also of the axones of the ventral cochlear nucleus, which wind over the latero-dorsal surface of the inferior cerebellar peduncle, pass medially beneath the ependyma of the floor of the fourth ventricle, as far as the median groove; and, crossing to the opposite side, sweep ventrally through the dorsal region of the medulla or pons, to join the tract of the lateral fillet, and so proceed in company with the other cochlear fibres to the higher levels. Not all the component fibres of the acoustic striæ follow the lateral fillet; some of them, after decussation, turn brainward, possibly joining the mesial fillet, whilst others may enter the posterior longitudinal fasciculus to assist in establishing the reflex paths, influencing the motor nerves. The auditory paths, by which impulses from the organ of Corti, travelling along the cochlear fibres reach the cerebral cortex, are as follows:

1. Peripheral neurones of the ganglion spirale whose axones—the cochlear fibres—pass to the reception nucleus, composed of the ventral and lateral cochlear nuclei.

2. The neurones of the cochlear nuclei send their axones—(a) By way of the corpus trapezoides to the superior olivary nucleus, chiefly of the opposite side or to the lateral fillet, or its nucleus without interruption in the olive. (b) By way of the striæ acusticæ through the tegmentum, to join the trapezoidal fibres.

3. The neurones of the superior olivary nucleus, or of the fillet nucleus, whose axones pass by way of the lateral fillet. (a) To the cells within the inferior colliculus, or (b) without interruption through the inferior brachium to the cells within the median geniculate body.

4. The neurones of the inferior colliculus and of the median geniculate body whose axones pass as the auditory radiation to the auditory cortical area within the temporal lobe of the cerebrum. The exact limitations of

the auditory area are still uncertain, but the most important part of it includes the superior temporal and the subjacent part of the middle temporal convolution.

The cochlear fibres that do not undergo decussation ascend through the lateral fillet of the same side, and eventually establish cortical relations with the corresponding cerebral hemisphere. From the foregoing it is manifest that the auditory area is chiefly connected with the cochlea of the opposite side. This crossed relation is emphasized, for there are sufficient reasons for asserting positively the existence of a similar crossed relation between the cerebellum and the semicircular canals and vestibule, as will appear later.

The fibres of the vestibular portion of the eighth nerve are the axones of the bipolar nerve cells situated within the small vestibular (g. vestibulare) or Scarpa's ganglion, which lies at the bottom of the internal auditory canal. The dendrites of these cells constitute the five branches of distribution of the vestibular nerve, and pass through the various openings in the inner wall of the bony labyrinth, as above described, to reach the maculæ acusticæ within the saccule and utricle and the cristæ acusticæ of the ampullæ of the semicircular canals, where the nerve filaments are in intimate relation with the neuro-epithelium. The centrally directed axones of the neurones supplying the utricle and the superior and external semicircular canals become consolidated to form the vestibular nerve of descriptive anatomy. Those from the saccule and posterior semicircular canal join the cochlear fibres, and with these course within the cochlear nerve until the latter unites with the vestibular to form the common auditory or eighth nerve trunk. Where the common trunk separates into its two roots the vestibular fibres leave the cochlear, and permanently assume their natural companionship with the remaining fibres of the vestibular root.

The vestibular fibres enter the brain stem at a slightly higher level than those of the cochlear root lying mesial to the latter and the ventral cochlear nucleus, and pass dorsally within the pons, between the inferior cerebellar peduncle and the spinal trigeminal root. On reaching a level dorsal to the latter, the vestibular fibres divide into short upward and longer downward coursing branches which, after condensing into an ascending and descending root respectively, end in arborizations around the cells of the vestibular nucleus of reception. The exact extent and constitution of this nucleus which underlies the area acustica in the floor of the fourth ventricle are uncertain, since the neurones directly related to the vestibular fibres contribute only a part of those contained within a large diffuse complex of cells and fibres, many of whose constituents probably have only an indirect connection with the vestibular nerve. Sabin[9] successfully reconstructed this complex, which comprises two general parts:

1. An extended, irregularly triangular mass of cells lying for the most part mesial to the tract formed by the ascending and descending branches of the vestibular fibres and,

2. A smaller mass of cells which lies above the larger one, and partly to the outer side of the tract of the vestibular fibres. The apex of the large triangular mass approaches the middle line, and its superior and inferior basal angles are prolonged upward and downward along the vestibular tract.

Examined microscopically the large mass is found to include three subdivisions:

(a) A tapering, caudally directed nucleus, which continues the inferior angle along the descending vestibular root.

(b) An extended triangular nucleus that includes the greater part of the large triangular mass.

(c) An irregular pyramidal nucleus that prolongs upward the superior angle. The first of these subdivisions (a) is known as the spinal vestibular nucleus (nuc. spinalis n. vestibularis); the second (b) as the median vestibular nucleus (nuc. medialis n. vestibularis), also as the chief nucleus or the triangular nucleus; and the third (c) as the superior vestibular nucleus, or the nucleus of v. Bechterew. The small mass (2) corresponds with the lateral vestibular nucleus (nuc. lateralis n. vestibularis), or the nucleus of Deiters.

The fibres of the descending root end around the neurones within the spinal nucleus in a manner similar to that in which the constituents of the spinal root of the trigeminus terminate in relation with the neurones within the substantia gelatinosa, whilst those of the ascending vestibular root end around the cells within the remaining vestibular nuclei. Ferrier and Turner, however, observed that after section of the eighth nerve the descending vestibular root did not degenerate, whilst Bruce traced the fibres to the cuneate nucleus. (See Vestibular connections.)

Although much uncertainty and conflict of opinion exist as to the details of the secondary paths by which the impulses carried by the vestibular fibres are distributed, it may be accepted that fibres pass from the nuclei of reception:

(a) To the cerebellum, chiefly to the roof nucleus of the opposite side, and possibly to the nucleus globosus and embolliformis as constituents of the nucleo-cerebellar tract, by which impulses of equilibration are carried to the great coordinating centres. (See note to Vestibular connections.)

(b) As arcuate fibres ventro-medially into the tegmentum of the pons across the middle line, bending upward or downward to reach other levels, some fibres, however, remaining on the same side. From the character of the impulses it is probable that only relatively few vestibular fibres join the median fillet to ascend to the optic thalamus. Other connections of the nuclei include:

(c) Commissural fibres between v. Bechterew's nucleus of either side.

(d) Fibres to the abducent nucleus. (But see summary of Vestibular connections.)

(e) Crossed and uncrossed fibres from Deiters' nucleus to the posterior longitudinal fasciculus; and

(f) Fibres from the same nucleus to the spinal cord.

Not all the neurones of Deiters' nucleus are concerned in transmitting afferent impulses to the cerebellum, for many are links in the path by which cerebellar cells exercise coordinating influence over the root cells of the spinal nerves. Starting in the cerebellum such efferent impulses are carried by fibres that descend through the median part of the inferior cerebellar peduncle, and probably end around certain of the cells within Deiters' nucleus.

From these cells in turn originate the fibres of the vestibulo-spinal tract which, after traversing the medulla, enter the antero-lateral column of the cord, and end in relation with the motor root cells. A shorter and more direct path for vestibular reflexes may probably be formed by the collaterals of the vestibular fibres that end around the spinal neurones of Deiters' nucleus; but the objection already put forward against the simple direct connection between afferent cochlear fibres and the oculo-motor centres holds as well here as in the case of vestibular fibres. Such connections cannot account for the more complex coordinated movements of equilibration which postulate a higher coordinating mechanism. The vestibular and cochlear nuclei are nutrient relay centres—coordinating centres of a lower order perhaps but related to, and acting in accord with, higher coordinating centres that regulate and control the more complicated movements consequent on cochlear or vestibular irritation. In those cases where there seems to be a direct connection between the vestibular fibres and the motor centres, e.g., those fibres mentioned as going to the abducent nucleus, it is probable that such fibres are mainly efferent and carry impulses from the cerebellum or other higher centres, which coordinate the movements of the eyes with the movements executed by other muscles in response to labyrinthine stimulation. The same has to be said of many fibres in the posterior longitudinal fasciculus, which, undoubtedly, convey coordinating impulses that harmonise the actions of motor nerve centres situated at some distance from each other, a notable instance being that of the abducens centre, which works in harmony with the centre for the internal rectus muscle of the opposite side. In the nystagmic movements observed in irrigations of the external auditory canal, undoubtedly the fibres from Deiters' nucleus to the posterior longitudinal fasciculus play an important rôle; but, as before stated, these fibres probably do not represent a direct immediate connection between the vestibular fibres and the posterior longitudinal fasciculus, but are to be considered in part at least as efferent fibres from the cerebellum or other coordinating centre.

In fishes the vestibular nerve is in close functional relation with the mesencephalic centres of the opposite side (Loeb[43]), whilst almost all the optic fibres pass to the same centres (Parsons[42]). In fishes, therefore, and in the lower forms of animals generally it is reasonable to assign important functions in the maintenance of equilibrium to the mesencephalic centres.

CHAPTER III

THE REMAINING CRANIAL NERVES

Of the remaining cranial nerves the first or olfactory is the only sensory nerve in man in which the neurones (cell bodies) of the first order are situated on the surface, like the sensory cells of some of the invertebrates. These latter cells represent the cell bodies of the posterior spinal ganglia, which have wandered to the surface epithelium (Retzius[10]). For the central relations of the olfactory nerve see "the rhinencephalon" in the synopsis of anatomical connections.

The second or optic nerve is not a true nerve, but a part of the brain substance. The true optic nerve components are the sensory neurones of the first order lying within the retina itself. As the dorsal-spinal-ganglion cells emigrate from the neural crest at an early age to form the neurones of the first order of the sensory spinal nerves, and lose their connection with the cord to regain it later on, it is manifest that the analogy between the optic and the sensory spinal elements is incomplete, since the retina develops entirely from the invaginated optic vesicle. For the central connections of the optic nerve see "Anatomical Synopsis."

The third, fourth, and sixth nerves are purely motor, and arise from their respective nuclei. As neuro-tendinous organs, similar to the Golgi-Mazzini corpuscles, have been demonstrated in the tendons of the external ocular muscles, and as the degree of convergence of the eyeballs has much to do with accommodation reflexes, it is probable that the third nerve has afferent paths for accommodation reflexes and for purposes of reciprocal innervation.

The nucleus of the third nerve is medially situated deep within the grey matter of the aqueduct of Sylvius, near the dorsal surface of the posterior longitudinal fasciculus. The nucleus is 6–8 mm long, and extends from the level of the caudal pole of the superior colliculi (from underneath superior colliculi and part of third ventricle, according to Quain[11]) almost to the level of the fourth nucleus, from which it is separated by a narrow interval. The grouping of the nerve cells of the third nucleus is as follows:

1. A paired group, consisting of long columns of cells—the chief nuclei—extending along the dorsal surface of the posterior longitudinal fasciculi. These chief nuclei are subdivided into (a) dorsal (lateral) and (b) ventral (mesial) cell groups. Dislocated groups of cells of the chief nucleus lie scattered among and beneath the fibres of the posterior longitudinal fasciculus.

2. Dorsal to the chief nucleus and overlying the posterior-median surface is the tapering column of small nerve cells—the Edinger-Westphal nucleus. The mass of this nucleus is thicker above than below (caudad), and is divided into dorso-lateral and ventro-median portions at the caudal pole, though fused at the cephalic extremity. There is question as to whether the Edinger-Westphal nucleus is related to the third nerve. In the dorsal (inferior) group of cells may be distinguished six secondary groups. Two of these lie lateral to the others, and somewhat dorsally. The remaining four are placed more mesially and one, which is in the middle line—the nucleus medialis—is common to the oculo-motor nerve of each side. Experiments on animals and pathological observations in man seem to show that the oculo-motor centres are grouped as above; that is, that they are divided into a superior and an inferior or dorsal group, and again into a mesial and a lateral group. The anterior (superior) part of the oculo-motor nucleus has smaller cells than the rest of the nucleus, and extends forward into the wall of the third ventricle. Hensen and Volckers[12] have shown by direct application of the electrodes in the dog that most anteriorily in the wall of the third ventricle is the centre for accommodation. Behind this, and more laterally situated than the former, is the centre for constriction of the iris. At the junction of the third ventricle with the aqueduct of Sylvius is the centre for the rectus internus, behind which, in order backward, are the

centres for the rectus superior, levator palpebræ superioris, rectus inferior, and lastly that of the obliquus superior. The clinical observations of Kahler and Pick, and those of Starr, indicate that the levator palpebræ, rectus superior, and obliquus inferior are innervated from the dorsal (dorso-lateral) group, and the rectus internus and rectus inferior from the ventro-mesial group in order from above downward (caudad).

v. Bechterew[13] from his observations following irritation of the posterior wall of the third ventricle and of the floor of the aqueduct of Sylvius concluded that the third nucleus consists of two larger groups—one paired and one unpaired—and of two smaller paired accessory groups. The former—larger groups—contain the main nucleus, and are laterally and ventrally in relation with the posterior longitudinal fasciculus. About the level of the junction of the middle with the anterior third of the nucleus is the median unpaired group and the dorso-lateral paired groups. The anterior (superior) groups are related to accommodation and pupillary constriction. The posterior group is subdivided into a lateral group for the levator palpebræ, superior rectus, and inferior oblique, and a median group for the internal and inferior rectus.

From the uniformity of the findings given above the grouping of the centres in the third nucleus may be accepted as proven. Tsuchida,[14] who recently (1906) investigated the whole subject, denies the constant existence of a well-marked, unpaired, median group, as described by Perlia and others, but admits the existence of broken groups of medially placed cells, especially in the upper and lower thirds of the nucleus. The oculomotor fibres, according to Tsuchida, originate from various portions of the third nucleus without limited relations to distinct groups.

The nucleus of Darkschweitsch, which is a laterally situated group of cells beginning in the floor of the third ventricle and extending caudally as far as the upper third of the chief nucleus, is no longer regarded as having direct relations with the third nerve. It is in intimate relation with the posterior longitudinal fasciculus. amongst the fibres of which its cells to a great extent lie.

Hence it is often called the nucleus of the posterior longitudinal fasciculus.

It is stated as probable that the oculo-motor fibres decussate in the caudal portion of the chief nucleus. Tsuchida[14] and others maintain that some decussation takes place throughout the greater part of the nucleus.

The connections of the third nucleus are as follows:

1. With the cerebral cortex: (a) With the posterior portion of the second and third frontal convolutions, chiefly of the opposite side. This connection is direct *via* the corona radiata, internal capsule, and cerebral peduncle, and was first demonstrated by Ferrier,[8] and independently by Munk. (b) With the occipital cortex about the visual area. This connection is also mainly crossed, and represents a motor ocular path, as demonstrated by Schäfer and Brown.[15] The path is indirect *via* the optic radiation, superior brachium, and superior colliculi. Ferrier also got ocular movements from this area, but considered them as associated movements (synkineses).

2. With the primary visual centres *via* the superior colliculi.

3. With the nuclei of the fourth and sixth cranial nerves and with Deiters' nucleus. These connections are supposed to be by way of the posterior longitudinal fasciculus.

4. With the nucleus of the facial nerve *via* the posterior longitudinal fasciculus. This connection is assumed to explain the associated action of the third and seventh nerves in contractions of the orbicularis palpebrarum and corrugator supercilii muscles.

As previously stated, the simple direct communication between the ocular nuclei and Deiters' nucleus cannot be accepted as fully explaining the coordinated actions of the various functionally related structures. Thus Duval and Laborde[30] showed that irritation of the sixth nucleus caused lateral conjugate movements of the eyeballs, and explained the results by asserting that the sixth nucleus sends fibres through the posterior longitudinal fasciculus to the centre for the opposite internal rectus in the third nucleus. But Schäfer[11] whilst admitting that fibres from the posterior longitudinal fasciculus enter the sixth and third nuclei, insists that such fibres end in these nuclei, and that no fibres from the sixth or third nucleus enter the posterior longitudinal fasciculus. The fibres of the posterior longitudinal fasciculus to the sixth nucleus more probably represent efferent cerebellar or other paths which, with similar fibres or collaterals passing from the cerebellum or other centre to the third and fourth nucleus, afford a more rational explanation of the coordinated action of these nuclei. Moreover, v. Bechterew[18] considers the cerebellar superior olivary tract which, through the superior olives, makes connection with the sixth nucleus as an efferent cerebellar path for ocular movements.

In a similar way, perhaps, may be explained the coordinated relation of the seventh and third, as well as of the other cranial nerve nuclei.

5. With the cerebellum. These paths have not been satisfactorily demonstrated. v. Bechterew states that a continuous connection exists between the third nucleus and the cerebellum, by which the latter becomes instructed as to the position of the eyeballs and the condition of the pupil. These pathways lie in the superior peduncle of the opposite side. The relation, therefore, between the oculo-motor nucleus and the cerebellum is a crossed one.

v. Bechterew,[18] however, in discussing the centrifugal function of the spinal bundle of the middle peduncle and of the cerebellar superior olivary tract, states that the fibres passing in the superior peduncle to the oculo-motor nucleus of the opposite side are also cerebello-efferent for the control of reflex ocular movements. The relations of the third nucleus to the seventh and to the other cranial nerve nuclei are probably mainly through the cerebellum *via* the superior peduncle, and thence through efferent cerebellar paths, including those in the posterior longitudinal fasciculus.

The fourth nucleus lies near the middle line, just caudad to the third nucleus, from which it is separated by a small interval. It seems to lie in a distinct depression on the posterior longitudinal fasciculus. It is 2 mm long, and extends from a point opposite the superior border of the inferior colliculus to the lower pole of the colliculus. It is in relation with the cortex of the inferior frontal convolution of the opposite side through fibres that pass by way of the corona radiata, internal capsule, and cerebral peduncle. It is stated that this nucleus is in relation with the third and sixth nuclei through the posterior longitudinal fasciculus. For reasons already stated, it is preferable to consider these relations as made mainly through the cerebellum or other coordinating centre, and not directly by fibres passing between the nuclei. The fibres arising in the nucleus pass laterally and ventrally in the tegmentum for a short distance, and then course in a median direction and dorsalward. They totally, or almost totally, decussate in passing through the anterior end of the superior medullary velum, and emerge from the surface of the latter at the side of the frenulum veli in two small bundles, which pierce the pia and unite to form the trunk of the nerve. The fourth nucleus does not seem to bear any close functional or anatomical relation to that of the seventh.

The sixth nucleus consists of multipolar cells lying in the dorsal part of the tegmentum of the pons, and underneath the floor of the fourth ventricle. It is situated anterior to the striæ acusticæ, beneath the eminentia teres, and ventral to and within the loop formed by the fibres on their way to form the seventh nerve. The sixth, like the third and fourth nuclei, is related to the frontal oculo-motor area of the cerebral cortex of the opposite side. The decussation of the cortical paths takes place at the level of the nucleus, and, according to some observers, through the nucleus. The nucleus, it is stated, is in relation with the third nucleus of the opposite side, and with the superior olive, and the cochlear and vestibular nuclei through the posterior longitudinal fasciculus. For reasons already stated, these connections probably represent in the main, efferent cerebellar paths, i.e., part of the common paths originating in the cerebellum, and sending fibres or collaterals to each of the associated nuclei. The sixth nerve makes its exit through the posterior part of the pons, between the latter and the medulla oblongata. In the cavernous sinus it is situated first above and then to the outer side of the carotid artery, and passes through the superior orbital fissure, to join the external rectus muscle. The sixth is the longest of the cranial nerves.

Owing to its length and the course it takes it is, of all the cranial nerves, the most liable to pressure from exudations, syphilitic or other, occurring at the base of the brain.

The fifth is a mixed nerve. It has two nuclei. The sensory nucleus is a columnar mass of grey matter within the lateral part of the tegmentum, extending from the middle of the pons through the entire length of the medulla, and into the spinal cord, as low as the second cervical segment, where it becomes continuous with the substantia gelatinosa. Though the rounded upper part of this tapering column is called the sensory nucleus of the fifth nerve, the whole mass receives fibres. The fibres of the fifth nerve as they approach this nucleus divide into ascending and descending bundles. The latter are coarser than the former, and form the descending or spinal root. The Gasserian ganglion contains the cell bodies of the primary neurones of the sensory part of the fifth nerve, this ganglion being the analogue of the dorsospinal-root ganglia of the spinal nerves.

The connections of the sensory part of the fifth nerve are:

1. By axones which cross the raphé from the nucleus to the opposite mesial fillet, ascend to the thalamus and, after interruption, proceed to the cortex cerebri. It is probable that some fibres pass to the fillet of the same side, and reach the brain as uncrossed paths. Collaterals of the crossed and uncrossed paths from the nucleus probably reach the motor nuclei of the fifth, seventh, ninth, and twelfth nerves of both sides. For reasons already stated it is probable that many of the functional relations between the cranial nuclei are mainly established through paths that pass by way of the cerebellum.

2. By axones from the cells of the fifth nucleus that enter the inferior peduncle and pass to the cerebellum as constituents of the nucleo-cerebellar tract. These paths are mainly crossed

3. By collaterals or stems of the arcuate paths with the sensory nuclei of the fifth, seventh, ninth, and tenth nerves of the opposite side.

The chief motor nucleus of the fifth (nucleus masticatorius) is a short column of grey matter lying in the upper part of the pons, close to the median side of the sensory nucleus.

The nucleus consists of large stellate cells, the axones of which pass through the tegmentum to the surface of the pons as the motor fibres of the fifth nerve. From the mesial portion of the nucleus a number of fibres pass dorsally in a curved manner toward the raphé, which they cross beneath the floor of the fourth ventricle to reach the opposite nucleus. A smaller constituent of the fifth motor root—the descending mesencephalic root—includes fibres that spring from cells lying within the lateral part of the grey matter in and about the Sylvian aqueduct. These fibres descend to join the larger tract from the chief motor nucleus. Some fibres that spring from the pigmented cells of the substantia ferruginea of the same and opposite side join the descending mesencephalic root of the fifth nerve.

The connections of the motor part of the fifth nerve are:

1. With the cortex of the lower third of the præcentral convolution of the opposite side mainly, but to some extent of the same side. The pathway is *via* the corona radiata, internal capsule, and cerebral peduncle into the pons, where, for the most part, the fibres decussate and end about the motor nucleus. The fibres from the cerebral cortex to the cranial motor nuclei, especially those to the seventh and twelfth, join the fillet, constituting what is known as the crustal fillet. (See Fillet.)

The seventh is now considered a mixed nerve. The cells of the primary sensory neurones of the seventh lie in the geniculate ganglion, situated in the knee of the facial canal and lying above the eighth and below the seventh nerve. The dendrites of these sensory neurones constitute the sensory peripheral fibres of the seventh, which course in the pars intermedia or nerve of Wrisberg. The axones of the geniculate neurones form the sensory root of the seventh. The sensory nucleus of reception of the seventh nerve is shared in common with the ninth and tenth nerves. The fibres of the seventh nerve on approaching this nucleus divide like those of the fifth into short ascending and long descending branches. They terminate about the neurones of the nucleus, the paths being continued by axones that pass to the mesial fillet of the opposite side, and eventually to the cerebral cortex. The afferent fibres of the seventh nerve are distributed chiefly through the chorda tympani, which emerges from the tympanic cavity through the Glásserian fissure, to join the lingual nerve. They mediate sensations of taste from the anterior two-thirds of the tongue. As these fibres end in part, at least, in the same nucleus of reception as the ninth nerve, they are considered by some authors as an aberrant portion of the latter. Because removal of the Gasserian ganglion has been followed by complete loss of taste in the corresponding side of the tongue (according to some surgeons in the anterior two-thirds only), it has been assumed that all the fibres of taste pass by way of the fifth nerve, or at least that those fibres of taste running in the chorda tympani belong in reality to the fifth nerve.

However, the results of Cushing,[19] following removal of the Gasserian ganglion, show that in no case was the sense of taste in the posterior third of the tongue affected, whilst that of the anterior two-thirds, though at first diminished or even lost, was subsequently completely restored. It seems, therefore, that the loss of taste following removal of the Gasserian ganglion was merely a bye-result of the operation. Cushing attributes the temporary loss of taste to post-operative degeneration and swelling of the fibres of the lingual nerve, which affected the conductivity of the intermingled fibres of the chorda tympani. The taste fibres of the chorda tympani, therefore, do not originate in the Gasserian ganglion, and the opinion given above referring their origin to the geniculate ganglion seems, if anything, to be substantiated. Hunt[20] has shown that inflammation of the geniculate ganglion is accompanied by herpes zoster of the tympanum, external auditory canal, and concha, often accompanied by peripheral facial palsy, tinnitus, deafness, and vertigo. The latter symptoms are due to the close relations of the seventh and eighth nerves within the temporal bone. Hunt further pointed out the association of inflammations of the Gasserian ganglion and the ganglia of the second, third, and fourth dorso-spinal cervical ganglia with herpes of the face and of the occipito-collaris region respectively, thus establishing the homology between the Gasserian and geniculate ganglia and those of the dorso-spinal roots. Head and Campbell [21] had previously shown that herpes zoster was due to a specific inflammation of the dorsal spinal root ganglia.

The motor nucleus of the seventh nerve lies in the tegmental region of the pons, ventral to the sixth nucleus, beneath the middle of the fourth ventricle. The connections of this nucleus are:

1. With the motor cerebral cortex of the opposite side by way of the corona radiata, internal capsule, crus cerebri, and the crustal fillet. The decussation takes place about the level of the nucleus of the pons.

2. With the nucleus of the opposite side. This connection has not been satisfactorily demonstrated.

From clinical and pathological observations it has been assumed that a special group of cells in the nucleus is related to the orbicularis palpebrarum and frontalis muscles. This group of cells is closely associated with the portion of the third nucleus innervating the levatores palpebrarum, and is bilaterally represented in the cerebral motor cortex. Hence the absence of paralysis of voluntary motion of the muscles that close the eye in hemiplegia from one-sided cerebral lesion, e.g., hemorrhage.

Ferrier [8] pointed out similar instances of bilateral cortical innervation by way of contrast with the one-sided cortical representation of speech in Broca's centre.

The seventh nerve supplies motor innervation to the muscles of the face, of part of the scalp, and of the ear, including its intrinsic muscles.

The secretory and vaso-dilator fibres (bulbar autonomics) for the submaxillary and sublingual salivary glands pass by way of the chorda tympani which, emerging from the tympanic cavity through the Glasserian fissure, joins the lingual nerve. After coursing a short distance with this nerve the secretory and vaso-dilator fibres for the salivary glands mentioned, branch off and pass to the glands along the ducts of the latter. The cells of the so-called submaxillary ganglion receive only fibres destined for the sublingual gland, as shown by Langley, who suggests that the ganglion be called the sublingual. The fibres to the submaxillary gland connect with nerve cells lying within the hilus of the gland. The sympathetic autonomics reach the glands by way of the superior cervical ganglion.

The ninth is also a mixed nerve. The cells of the primary sensory neurones lie in the superior and petrous ganglia (g. superius and g. petrosum), situated upon the upper part of the nerve trunk as it emerges from the skull. The sensory root traverses the formatio reticularis grisea, and just before reaching the dorsal nucleus divides into a mesial and a lateral bundle of fibres. The former and smaller bundle ends about the cells of the sensory part of the dorsal nucleus (nuc. alæ cinereæ). It is supposed that the cells in the upper part of the nucleus are related to the sense of taste, and it is about these cells that the taste fibres of the chorda tympani end in part at least. The second and larger bundle of fibres forms the chief constituent of the fasciculus solitarius along the surface and between the fibres of which lies a slender column of grey matter. The cells of this grey matter constitute the spinal reception nucleus of this part of the ninth nerve.

The motor portion of the ninth nerve springs from cells lying in the dorsal nucleus and in the nucleus ambiguus. It is in relation with the cortex of the lower part of the præcentral gyrus of the opposite side. The ninth nerve mediates sensations from the mucous membrane of the tongue and pharynx (in part), and from the tympanic cavity and Eustachian tube. It sends motor fibres to the muscles of the pharynx and base of the tongue and secretory fibres to the parotid gland. The dorsal nucleus, which is the nucleus of origin and reception for the ninth and tenth nerves, and for the taste fibres of the chorda tympani passing in the seventh, lies just lateral to the median vestibular and upper part of the twelfth nucleus. Its upper portion is covered by the spinal vestibular nucleus and its lower portion overlies the twelfth nucleus. The middle portion of the nucleus—the fovea vagi—is in intimate relation with the floor of the fourth ventricle. The median portion of the nucleus constitutes the dorsal motor nucleus and the remaining portion the dorsal sensory nucleus. The nucleus ambiguus (nucleus ventralis) lies in the formatio reticularis grisea, midway between the substantia gelatinosa and the dorsal accessory olivary nucleus. It consists of a small group of cells, giving origin to motor fibres, which unite with the fibres from the dorsal motor nucleus. The motor nuclei of the ninth are in crossed relation with the cerebral cortex, like the other cranial motor nerves.

The sensory part of the dorsal nucleus (nuc. alæ cinereæ) and the spinal nucleus (nuc. tractus solitarii), which resembles the corresponding nucleus of the fifth nerve, are reception nuclei for the lateral mixed nerves (VII, IX, and X). The fasciculus solitarius extends from the upper border of the medulla to the lower limit of the decussation of the fillet, and is related to the sensory fibres of the seventh, ninth, and tenth nerves. The largest part of this tract consists of the root fibres of the ninth nerve; in fact, the tract itself is in reality only the continuation of the larger bundle of fibres of the ninth nerve on its way to join its nucleus of reception, the latter consising of cells lying upon and within the fasciculus solitarius.

The tenth (vagus, pneumogastricus) is a mixed nerve. The cell bodies of the primary sensory neurones of the vagus lie within the ganglion of the root in the jugular foramen and the ganglion of the trunk outside the jugular foramen, both ganglia being situated upon the upper part of the nerve.

The centrally directed processes pass into the medulla in company with the motor strands, and divide into two sets of fibres, of which the larger set ends about the lower portion of the dorsal sensory nucleus, whilst the smaller set bends downward and enters the fasciculus solitarius to end about the cells of the reception nucleus, situated upon and between the fibres of the fasciculus. The sensory fibres, therefore, of the ninth and tenth nerves, as well as part, at least, of the seventh (chorda tympani fibres) have a more or less common nucleus of reception in the sensory part of the dorsal nucleus (nuc. alæ cinereæ) and in the spinal reception nucleus of the fasciculus solitarius (nuc. tractus solitarii).

The central connections of the vagus, like those of the other mixed nerves, include:

1. Fibres to the median fillet, chiefly of the opposite

side, which pass to the optic thalamus. From the latter they are relayed to the cerebral cortex.

2. Fibres that pass to the cerebellum, mainly of the opposite side, as part of the nucleo-cerebellar tract. Ross[22] considered the cells of the sensory part of the vagus as the analogues of the vesicular cells of Clarke's column.

3. Fibres to various other nuclei and centres in the medulla, such as the vaso-motor and respiratory centres.

The sensory portion of the vagus mediates sensation from the mucous membrane of the pharynx (branch from the ganglion of the trunk), larynx, trachea, œsophagus, stomach, intestines, gall-bladder, gall-ducts, and from the lungs and pericardium. The auricular branch from the ganglion of the root supplies the skin of the posterior portion of the auricle and the posterior inferior portion of the external auditory canal.

Besides the above there are special fibres to the heart, liver, spleen, pancreas, kidneys, adrenals, and possibly to the intestinal blood-vessels.

The bulbar or accessory portion of the eleventh nerve is generally admitted to be an integral part of the motor division of the tenth. It is still a question as to the ultimate distribution of these fibres as well as to that of some of the motor fibres of the tenth nerve proper.

Van Gehucten[23] believes that the accessory fibres are distributed chiefly to the larynx *via* the inferior laryngeal nerve, a branch of the tenth, and do not reach the heart or stomach. Brunton[24] concluded that the inhibitory cardiac fibres originate in the accessory nucleus. The efferent fibres to the heart from the tenth nerve proper are most probably inhibitory, and it is still a question whether they reach the heart muscle directly or end about neurones within the heart structure.

The efferent fibres of the tenth to the stomach and intestines include motor, inhibitory, and secretory fibres. It is stated that some of the fibres affect the calibre of the blood-vessels of the stomach and liver, but Burton-Opitz[44] has shown that no vaso-motors for the stomach or liver run in the pneumogastric nerve. The motor fibres of the tenth nerve originate in the cells of the motor portion of the dorsal nucleus, and in those of the nucleus ambiguus, more or less in common with the motor fibres of the ninth nerve. The accessory fibres spring from cells located within the nucleus ambiguus only. The fibres from the nucleus ambiguus at first pass backward toward the floor of the fourth ventricle, and then bend sharply outward, and become condensed into compact strands that receive motor fibres from the dorsal nucleus. From this point they proceed ventro-laterally with the sensory fibres to the superficial origin of the nerve.

The eleventh or accessory nerve is a purely motor nerve supplying the sterno-mastoid and trapezius muscles. Its upper medullary roots spring from the cells of the motor portion of the dorsal nucleus, which gives origin to the vagus, whilst its lower, spinal roots spring from the anterior horn of the grey matter of the cord, as low as the fifth or seventh segment. The relation of the upper portion of this nerve to the vagus, of which it is considered to be a part, has already been discussed.

The twelfth or hypoglossal is also a pure motor nerve supplying the muscles of the tongue, the extrinsic muscles of the larynx, and those attached to the hyoid bone. Its fibres originate in a distinct nucleus lying in the floor of the fourth ventricle near the middle line.

The eleventh and twelfth nerves, like the other motor cranial nerves, are related to the cerebral cortex of the opposite side. They are also related to the other cranial nuclei to some extent through the cerebellum. It is stated that the third, seventh, and twelfth nuclei are in intimate relation. The objections urged against the simple direct relations between the cranial nuclei render it probable that the chief relation between these three nuclei is an indirect one through the medium of a common coordinating centre, such as the cerebellum.

Gaskell[25] has shown that every spinal segment has a somatic and a splanchnic root. Each somatic root is divided into a ganglionated and a non-ganglionated portion springing from the cells of the posterior (dorsal-spinal-root.ganglion) and anterior horns respectively. The splanchnic root is also divided into ganglionated and non-ganglionated portions, which spring from the cells of the lateral horn and those of Clarke's column respectively. The ganglia of the splanchnic root are the ganglia of the sympathetic chain, and are the homologues of the ganglion trunci vagi. Thus in the eleventh nerve the somatic root, besides sending a branch to the cervical plexus, is distributed to the sterno-mastoid and trapezius muscles, whilst the splanchnic root from the lateral horn is distributed to the viscera.

CHAPTER IV

BLOOD-SUPPLY OF THE LABYRINTH AND OF THE CRANIAL NUCLEI

The labyrinth is supplied by the internal auditory artery, a branch of the basilar, which accompanies the auditory nerve. This vessel supplies the vestibule, semicircular canals, cochlea, and their membranous contents. The internal auditory divides into the common cochlear (Siebenmann) and the internal vestibular artery. The latter vessel supplies the vestibule. The former divides into two branches, one of which, the cochlear artery proper, supplies the two superior spirals of the cochlea, whilst the other—the vestibulo-cochlear artery—supplies the inferior spiral of the cochlea and the vestibule. The blood is returned by the internal auditory vein into the superior petrosal sinus, and by small veins which pass through the cochlear and vestibular aqueducts to the inferior and superior petrosal sinuses. Siebenmann found that the veins from all the semicircular canals united in a common trunk, which leaves the labyrinth along the aquæductus vestibuli; but Shambaugh[7] found that in the sheep, calf, and pig the venous blood from the vestibule and semicircular canals drained into the canaliculi cochleæ, but that in the calf the vein from the crus commune often leaves the labyrinth along the aquæductus vestibuli.

The labyrinthine artery is thus a part of the central brain system, and susceptible of similar pathological changes.

The branches to the cochlea, vestibule, and semicircular canals form delicate convolutions in intimate relation with the cochlear and vestibular nerve elements. Witmaack,[26] discussing the causation of senile deafness, attributes the latter to disturbed nutrition of the cochlear nerve, caused by arterio-sclerosis. The "elective vulnerability" of the cochlear branch of the auditory nerve is due, in part, to the fact that this nerve is imbedded in a bony capsule, partly surrounded by broad, lymphatic spaces. The blood-vessels are end arteries, whilst the ganglion cells of the cochlear ganglion are bipolar, and are much smaller than all other ganglion cells found in vertebrates.

The vessels of the labyrinth are not affected by intracranial pressure like those of the retina, because most of the venous blood and lymph passes from the aquæductus cochleæ into the jugular vein. In conditions like tumor of the brain the ear presents no sign analogous to the retinal choked disc.

The region of the third nucleus is supplied by small delicate branches of the posterior cerebral artery. These branches are end arteries; that is, they form no anastomoses, and have no communication with the vascular system of bordering regions. Moreover, these branches change from a horizontal to a vertical course, and they, as well as the superior and middle cerebellar arteries (branches of the basilar) originate at a point where the carotid and basilar blood-streams meet. These facts have been adduced to explain the momentary nystagmus, vertigo and disturbances of vision which attend sudden stooping and rising to the erect position, the chief factor in the causation of these symptoms being sudden alteration in the blood-supply of the ocular, cerebellar, and other centres.

The pons and medulla are supplied by the vertebral arteries and by branches springing from them, viz., the basilar and anterior spinal. The latter supplies the medulla oblongata and the nuclei situated in its lower region, including the eleventh and twelfth. The fifth, sixth, and seventh nuclei are supplied by branches of the basilar. The medulla oblongata receives further blood-supply from branches of the dorsal spinal, posterior cerebral, and inferior and posterior cerebellar arteries. The central artery of the medulla, a branch of the basilar, sends delicate branches to the cochlear and vestibular nuclei. A branch of the posterior communicating artery furnishes a terminal to the red nucleus. The vermis of the cerebellum receives a branch from the superior cerebellar artery, whilst the nucleus dentatus is supplied by a branch from the middle cerebellar. The cerebral peduncle, through which pass the bulbo-cerebral and cerebello-cerebral paths receives branches from the posterior cerebral arteries. Branches from the posterior communicating and from the anterior choroid arteries reach the internal capsule.

It is stated (Kyle[37]) that affections tending to obstruct the vessels supplying the cortical centres and the nuclei and paths related to the vestibular system may cause vertigo and other symptoms usually referred to disturbances within the labyrinth.

In general, it may be said that the vascular supply of the base of the brain is faulty from the standpoint of collateral circulation. Hence the persistence of symptoms from small vascular lesions of areas adjoining the base, lesions which, if they occurred upon the convexity of the hemispheres, would easily be compensated by collateral blood-supply. The area of the third nucleus having the poorest blood-supply of any part of the brain, every serious disturbance of the circulation has its effect upon the innervation of the ocular muscles. Thus in profound anæmic states from loss of blood or other cause, and in the cachexia of wasting diseases, it is common to find disturbances of vision, some nystagmus, and vertigo. These symptoms are similarly encountered when a person who has lain in bed some time first assumes the erect posture. Here the vaso-motor mechanisms have become disordered from disuse, and there ensue, when the patient first attempts to stand up, temporary irregularities in the blood-supply of the ocular and possibly other centres.

CHAPTER V

SYNOPSIS OF ANATOMICAL CONNECTIONS

Connections of the Vestibular Nerve

The vestibular nerve ends in the vestibular nuclei of reception. From the nuclei fibres pass:

(a) To the cerebellar roof nucleus of the opposite side as the vestibulo-cerebellar tract. Risien-Russell[28] and Ferrier and Turner[29] showed that this is an efferent path between the vermis and Deiters' nucleus, but Kölliker, from embryological studies, concludes that some of the fibres are axones of Deiters' cells a few coming from the chief auditory nucleus.

(b) As arcuate fibres passing ventro-medially into the tegmentum of the pons to cross the middle line, and so bend upward and downward to reach other levels. Some fibres, however, remain on the same side. Of the crossed fibres some are said to join the mesial fillet to reach the thalamus and superior colliculi, while others are said to join the lateral fillet and so reach the inferior colliculi, and possibly the auditory cortex.

(c) Commissural fibres between the nucleus of Bechterew or superior vestibular nucleus on either side.

(d) Fibres to the abducent nucleus. Gordinier and others believe that each vestibular nucleus sends fibres to the abducent nucleus on either side. Duval and Laborde showed that the sixth nucleus was connected with the third and fourth nerve nuclei of the opposite side, and stimulation of the area of the sixth nucleus caused the eyes to turn to the side of stimulation. This, however, does not prove that the sixth nucleus coordinates these movements. Schäfer insists that no fibres enter the posterior longitudinal fasciculus from the sixth or third nucleus. Moreover, it is not apparent why the fibres to the sixth nucleus are necessarily the direct continuations of vestibular fibre-paths, and not the continuation of descending paths from the cerebellum. The posterior longitudinal fasciculus is in intimate relation with the nuclei of the sixth, third, and fourth nerves, and receives numerous fibres, both crossed and uncrossed, from the vestibular (including Deiters') and from other nuclei. Moreover, when eye movements take place in response to vestibular stimulation movements of other parts take place concurrently and in a coordinated manner. Hence but little importance is to be attached to this connection between the vestibular nerve and the sixth nucleus, unless it be distinctly understood as an indirect connection through the medium of the cerebellum or other coordinating centre *via* the vestibular (Deiters') nucleus or the posterior longitudinal fasciculus, or both.

(e) Crossed and uncrossed fibres to the posterior longitudinal fasciculus.

(f) Fibres to the spinal cord. These fibres communicate with the olivary body and lateral columns. The descending vestibulo-olivary tract passes to the olivary body of the same side (Van Giesen[31]), whilst the vestibulo-spinal tract passes through the periphery of the lateral field of the formatio reticularis, and descends in the lateral column of the cord. Its ultimate distribution is unknown, but it probably ends in relation with the ventral horn cells.

The fibres to the spinal cord and superior olive are not altogether to be considered as mainly continuations of paths direct from the vestibular nerve. For the reasons given in discussing the fibres to the sixth nucleus these fibres are to be regarded as coming in part from the cerebellum *via* Deiters' nucleus. The latter has many afferent and efferent neurones, and is, therefore, properly considered as a relay station for both afferent and efferent impulses, rather than a coordinating centre of the higher order. More recent investigations show that part of the ventral (anterior) marginal fasciculus (vestibulo-spinal tract) comes from the roof nucleus of the cerebellum (Morris[32]). The same author states that the fibres from the vestibular nucleus to the abducent nucleus travel by way of the posterior median (longitudinal) fasciculus of both sides.

The vestibulo-olivary tract is best regarded as being in part at least the continuation of vestibular paths after interruption in Deiters' nucleus. Reaching the olive on the same side the fibres cross to the opposite olive and proceed in part to the cerebellum by way of the restiform body. This view seems to be supported by v. Bechterew's experiments, in which lesions of the inferior olive caused rotations and ocular deviations, exactly like those following section of the vestibular nerve on the same side. The tract may, therefore, be called the crossed vestibulo-olivo-cerebellar tract, whilst the descending vestibulo-spinal tract may be called the cerebello-vestibulo-spinal tract. The olivary fasciculus, or Helweg's bundle, connects the spinal neurones with the olive and indirectly with the cerebellum (Morris).

Much confusion is created by anatomical writers retaining the term, "direct sensory cerebellar tract of Edinger." This tract was described by Edinger in his earlier work, viz., "Twelve Lectures on the Structure of the Central Nervous System." In his later work, "The Anatomy of the Central Nervous System of Man and of the Vertebrates in General," Edinger himself discards the term, "direct sensory cerebellar tract," saying: "It is wiser at present to designate the system as tractus cerebellaris acustica, etc." We may, therefore, conclude that the direct sensory cerebellar tract contains nothing more by way of afferent paths than what is included in the sensory nucleo-cerebellar paths (q.v.).

According to Cunningham,[33] both the principal nucleus and the nucleus of Deiters are in intimate relation with the superior worm of the cerebellum. Deiters himself, as well as Ferrier and Turner, believe that Deiters' nucleus is an internode between the cerebellum and the cord. Ferrier and Turner adduce strong evidence to support this view. But Kölliker has shown that some of the paths are afferent, i.e., to the cerebellum. Klemoff[34] believes the axones of Dieters' nucleus form the ventral (anterior) tract of Löwenthal, i.e., the tractus vestibulo-spinalis of Monakow, descending in the anterolateral column of the cord.

Ferrier and Turner observed that after section of the eighth nerve, the descending vestibular root did not degenerate. They believe, therefore, that this root forms an internodal connection between Deiters' nucleus and the cuneate nucleus. Bruce saw the lower end of the descending vestibular root terminate in the cuneate nucleus. It seems probable, therefore, that the descending vestibular root is a continuation of afferent cerebellar paths represented in the dorsal fasciculi (columns of Goll and Burdach).

Cerebellar Connections

1. Through the inferior peduncle by:

(a) The direct cerebellar tract (dorsal spino-cerebellar fasciculus or tract of Flechsig) continues, after interruption, the afferent paths from the posterior nerve roots of the same side to the superior worm of the same and opposite side, i.e., in the monticulate and lingual lobules.

(b) The arcuate fibres—anterior and posterior superficial—from the gracile and cuneate nuclei of the same and opposite side. The fibres of the so-called descending root of the vestibular nerve may be grouped here. (See Vestibular connections.)

(c) The olivo-cerebellar fibres, chiefly from the opposite inferior olivary nucleus, but to a limited extent from the nucleus of the same side. These constitute the bulk of the latter part of the restiform body, and end within the cortex of the hemisphere and worm, and also within the fibre complex enveloping the nucleus dentatus. The fibres are mostly afferent (v. Bechterew[18]). The atrophy, however, of this nucleus, which is present in failure of development of the opposite half of the cerebellum or following its removal, seems to show that many of the cerebello-olivary fibres are efferent paths. The further links of connection are uncertain.

Kölliker maintains that some of the olivo-cerebellar fibres are axones of the cells of Purkinje from the opposite side, and that fibres from some olivary cells pass downward into the anterior ground bundle. The possibility of some of the olivo-cerebellar fibres being continuations of vestibular afferent paths should be remembered. The route in such a case would be along the vestibular nerve to Deiters' nucleus, thence to the olive on the same side, thence cross the midline to the opposite olive and through the restiform body to the cerebellum. Such a connection would explain the phenomena observed by v. Bechterew[13] following destruction of the olive and many other experimental phenomena which otherwise are not readily explicable.

From the manifestly important connections between the olive and the cerebellum it would seem that the olive is a great relay station for afferent and efferent cerebellar paths, if not a coordinating centre of equilibrium on its own account. v. Bechterew believes the inferior olive is related closely to tactile sensibility.

(d) Fibres from the nucleus lateralis of the medulla to the cortex of the cerebellar hemisphere. The nucleus lateralis is a collection of cells in the formatio reticularis grisea near the periphery and ventral to the spinal trigeminal root. The nucleus is regarded as the analogue of the lateral horn cells.

(e) Fibres from the arcuate nucleus (a collection of cells in the paths of the anterior superficial arcuate fibres) to the cerebellar cortex.

(f) The nucleo-cerebellar tract, i.e., fibres from the reception nuclei of the trifacial, facial, vestibular, cochlear, glosso-pharyngeal, and vagus. This tract occupies the middle of the peduncle, and ends chiefly in the roof nucleus (nucleus fastigii) of the same and opposite side. This tract includes the direct sensory cerebellar tract of Edinger. (See Vestibular connections.)

(g) Fibres from the cerebellar roof nucleus to Deiters' nucleus, and thence by the vestibulo-spinal tract to the antero-lateral column of the cord (ventral marginal fasciculus and the fasciculus from the formatio reticularis).

(h) According to some anatomical writers, additional vestibular, and possibly other sensory fibres, pass without interruption by way of the restiform body to the cerebellar roof nuclei, constituting the direct sensory cerebellar tract of Edinger. As before noted, Edinger's direct sensory cerebellar tract, considered as an afferent cerebellar pathway, is in reality identical with the nucleo-cerebellar tract.

Connections of the Cerebellum Through the Middle Peduncle

Afferent paths, i.e., to the cerebellum.

1. Fibres from the cells of the pontine nucleus as continuations of the fronto-cerebellar and of the temporo-occipito-cerebellar tracts. These paths connect the cells of the frontal, temporal, and occipital lobes with the cortex of the opposite half of the cerebellum, ending chiefly in the cortex of the hemisphere, in the worm, and possibly in the nucleus dentatus. The tracts are chiefly crossed.
2. Collaterals from pyramidal tracts to the pontine nuclei.

Efferent paths, i.e., from the cerebellum.

1. Fibres—the axones of the Purkinje cells in the cerebellar cortex—which cross the middle line to end in relation with the cells of the nucleus tegmenti (red nucleus), close to the raphé (v. Bechterew[18]). The tract is mainly uncrossed, owing to a double decussation, viz., in the superior peduncles and in Forel's decussation.
2. Fibres from the cells of Purkinje to the nucleus pontis of the same and opposite side have been assumed to exist. Ferrier maintains that the cerebello-pontine fibres of the middle peduncle, after crossing the middle line, join the pyramidal tract, to recross in the pyramidal decussation. Such a pathway would establish efferent relations between each half of the cerebellum and the structures of the same side of the body. Ferrier's experiments undoubtedly establish a marked functional relationship between these parts, but recent observations seem to show that the efferent cerebello-pontine fibres do not end in the nucleus pontis but, after crossing the middle line, are believed to end within the tegmentum in the red nucleus (v. Bechterew).

Ferrier, however, makes no mention of the nucleus pontis, nor of interruption of the efferent paths at this point. v. Bechterew's assumption may be accepted, and there is still provision for a possible recrossing of this efferent cerebellar path by fibres which emerge from the ventro-medial surface of the red nucleus, and cross the side, thence cross the midline to the opposite olive and through the restiform body to the cerebellum. Such a connection would explain the phenomena observed by v. Bechterew[12] following destruction of the olive and many other experimental phenomena which otherwise are not readily explicable.

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1. Fibres—the axones of the Purkinje cells in the cerebellar cortex—which cross the middle line to end in relation with the cells of the nucleus tegmenti (red nucleus), close to the raphé (v. Bechterew[13]). The tract is mainly uncrossed, owing to a double decussation, viz., in the superior peduncles and in Forel's decussation.

2. Fibres from the cells of Purkinje to the nucleus pontis of the same and opposite side have been assumed to exist. Ferrier maintains that the cerebello-pontine fibres of the middle peduncle, after crossing the middle line, join the pyramidal tract, to recross in the pyramidal decussation. Such a pathway would establish efferent relations between each half of the cerebellum and the structures of the same side of the body. Ferrier's experiments undoubtedly establish a marked functional relationship between these parts, but recent observations seem to show that the efferent cerebello-pontine fibres do not end in the nucleus pontis but, after crossing the middle line, are believed to end within the tegmentum in the red nucleus (v. Bechterew).

Ferrier, however, makes no mention of the nucleus pontis, nor of interruption of the efferent paths at this point. v. Bechterew's assumption may be accepted, and there is still provision for a possible recrossing of this efferent cerebellar path by fibres which emerge from the ventro-medial surface of the red nucleus, and cross the middle line as the decussation of Forel bending downward as the rubro-spinal tract.

The middle cerebellar peduncle, according to v. Bechterew, contains: (a) Cerebral fibres which originate in the superior and lateral parts of the cerebellar cortex, and in the area of the upper worm and central nucleus, and pass to the upper half of the pons, where some of the fibres from the cerebrum along the medial and lateral divisions of the cerebellar peduncles are interrupted. The motor tracts of the cerebrum are joined in the ganglia of the pons by impulses from the cerebellum conveyed in the middle peduncle.

(b) The spinal fibres, which are cerebello-fugal, arise from the anterior and middle parts of the cerebellar cortex, and end in the grey matter of the lower half of the pons on both sides. From this point fibres pass as the fasciculus verticalis to the region of the nucleus reticularis and the lateral area of the formatio reticularis, and finally to the antero-lateral ground bundles of the cord (fasciculus to and from the formatio reticularis). A few fibres reach the corpora quadrigemina with the lemniscus. From the above it is manifest that v. Bechterew agrees with Ferrier in the main facts.

2. Fibres to the corpora quadrigemina *via* the spinal bundle and the median fillet (v. Bechterew).

Connections of the Cerebellum Through the Superior Peduncle

Afferent paths, i.e., to the cerebellum.

1. Fibres from Gowers' tract (ventral spino-cerebellar tract), arching over the fifth nerve to reach the superior peduncle and thence to reach the dentate nucleus and cerebellar cortex of the same side (Hoche). As some of the posterior root fibres, of which Gowers' tract is a continuation, decussate in the anterior commissure, this tract is, in part, crossed.

Mott believes the fibres of Gowers' tract taking this course end in the posterior portion of the vermis, whilst the remaining fibres of Gowers' tract end in the corpora quadrigemina and the thalamus.

2. Fibres from the red nucleus to the dentate nucleus. This path is mainly crossed.

3. Fibres to the cerebellum from the oculo-motor nucleus—afferent ocular and pupillary paths (v. Bechterew). This path is crossed.

Efferent paths, i.e., from the cerebellum.

1. Fibres of the cerebello-tegmental tract, which spring from the dentate nucleus with probably augmentations from the roof nucleus and cortex of the worm. At the decussation of the superior peduncles most of the fibres cross. Above this decussation the fibres are in large measure interrupted in the red nucleus, those not so interrupted passing through the subthalamic region to end in relation with the cells of the thalamus. Most of the fibre-paths ending in the red nucleus are continued by rubral neurones to the thalamus. From the thalamus the uninterrupted paths, as well as those interrupted in the red nucleus, are continued to the cerebral cortex by the thalamo-cortical paths. Many of the fibre-paths ending in the red nucleus are, however, diverted into the rubrospinal tract which, for the most part, crosses the median line in the decussation of Forel, and traverses the brain stem and antero-lateral columns of the cord to reach the ventral root cells. The connections of the cerebellum through the rubro-spinal tract are, therefore, chiefly uncrossed.

The axones of the red nucleus neurones, which form the rubro-spinal tract, emerge from the ventro-medial surface of the nucleus, cross the middle line at the decussation of Forel, and bend downward within the tegmentum of the mid-brain and pons to reach the medulla, finally entering the lateral columns of the cord as an important but uncertainly defined descending tract, which may be fairly represented as the intermediate fasciculus. The cerebello-rubro-thalamico-cortical paths are mainly crossed, while the cerebello-rubro-spinal tracts, owing to a double decussation, bring the cerebellum into relation mainly with the motor root cells of the same side.

2. Fibres to the oculo-motor nucleus of the opposite side. These fibres contain the afferent pupil reflex path (v. Bechterew[13]), but they also contain efferent paths,

probably concerned in the conjugate movements of the eyes. This tract is crossed.

The dorsal zones of the early metencephalon besides providing the reception nuclei of the sensory cranial nerves, and perhaps of the pontine nuclei, contribute the neuroblasts, which become the nervous elements of the cerebellum. The ventral zones play an active part in producing the tegmental portion of the pons and nuclei of origin of the fifth, sixth, and seventh cranial nerves. As in the medulla so in the pons, the ventral tracts are relatively late additions to the tegmentum, which is the primary and oldest part of this segment of the brain stem. The bulky ventral nervous masses take form only after the appearance of the cerebro-spinal and cerebro-cerebellar paths. The human cerebellum is, therefore, developed from the roof-plate and adjacent parts of the dorsal zones of the lateral walls of the metencephalon.

The posterior longitudinal fasciculus contains:

1. Fibres from the nucleus of the posterior commissure (Darkschewitsch's nucleus) in advance of the third nerve nucleus in the grey matter, about the upper end of the Sylvian aqueduct. The fibres cross to join the fasciculus of the opposite side.

2. Fibres, also crossed, from the nucleus fasciculi longitudinalis dorsalis in the grey matter of the floor of the third ventricle in the vicinity of the corpus mammillare.

3. Fibres crossed and uncrossed from the vestibular (Deiters') nucleus. For reasons already stated in discussing the vestibular connections, it is highly improbable that the paths lying between Deiters' nucleus and the posterior longitudinal fasciculus merely represent the continuations of paths from the semicircular canals to Deiters' nucleus, and thence to the ocular and other nuclei. Many of the fibres undoubtedly are continuations of paths from the cerebellum and other higher centres, perhaps, and carry efferent impulses, whilst others are afferent—the continuations perhaps of vestibular paths, but their main objective point is not the sixth nucleus, but rather some higher coordinating centre. Either of these suppositions makes it possible to explain the phenomena observed on stimulating the area corresponding to the location of the

sixth nerve nucleus without according this latter centre the dignity of a coordinating centre for the other ocular muscles, and also without encountering the awkward objections the latter hypothesis entails.

4. Fibres from the abducens nucleus to the oculomotor nucleus. The existence of these or of any fibres leaving the abducens nucleus to enter the posterior longitudinal fasciculus is denied by Schäfer.[11]

5. Fibres from the reception nuclei of the remaining sensory nerves of the brain stem of the same and opposite sides. These fibres are probably all afferent, their objective point being the higher coordinating centres, and perhaps also centres at other levels, which by their means —through the posterior longitudinal fasciculus—may be brought into relation for reflex movements of a lower order.

6. Fibres traversing the commissura hypothalamica (Edinger[35]), to reach probably the mammillary body of the opposite side.

7. Fibres probably also to the thalamus, subthalamic region, the corpora quadrigemina, the red nucleus, and the cerebellum.

The connections of the posterior longitudinal fasciculus are important and far-reaching, but they have been only imperfectly worked out. The connection with the cerebellum is undoubted, and yet no definite path of any importance has been traced. The same has to be said of its connections with other higher centres.

Connections of the Mesial Fillet or Lemniscus

1. Fibres from the nucleus gracilis and nucleus cuneatus which, for the most part, cross the middle line as the arcuate fibres making the sensory decussation about the upper border of the pyramidal decussation in the lower part of the medulla oblongata. This sensory decussation marks the lowest limit of the fillet and the fibres composing it are continuations of the paths represented in the posterior fasciculi of the cord, viz., those of Goll and Burdach derived from the posterior nerve roots of the same side. The fillet tract is mainly crossed.

2. Fibres from the reception nuclei of all the sensory

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cranial nerves connected with the brain stem. These fibres are mainly crossed.

3. Fibres from the cells of the more extensive nuclei, e.g., from those of the substantia gelatinosa. Accompanying the spinal root of the fifth nerve, numerous fibres sweep toward the raphé and, with few exceptions, cross to join the fillet of the opposite side.

These three sets of fibres constituting the bulbo-tecto-thalamic tract course upward through the tegmentum. Many of the fibres end around the deeper grey stratum of the superior colliculus, some passing over the aqueduct of Sylvius to the opposite colliculus. The remaining fibres pass on to reach the cells in the ventral part of the optic thalamus, thence the paths are continued to various parts of the cerebral cortex. Other fibres said to be derived from the cuneate nucleus end in the corpus subthalamicum and the lenticular nucleus. From the cells of the latter, fibres proceed through the commissure of Meynert —a strand placed just above the optic chiasm—to the lenticular nucleus of the opposite side. Still other fibres can be traced into the posterior commissure of the brain and into the mammillary body.

The fillet has also strands running in an opposite direction. Some of these are probably:

4. Fibres from the cells of the optic thalamus and corpora quadrigemina.

5. Efferent strands which establish connections between the cerebral cortex and the nuclei of the motor cranial nerves, especially the seventh and twelfth. These cortico-bulbar tracts join the median fillet in the upper part of the pons, and descend with it as far as the upper limit of the twelfth nucleus. This part is often called the crustal fillet. On reaching the levels of the various nuclei, the fibres destined for them undergo decussation for the most part.

6. Fibres from the cerebellum *via* the spinal bundle of the middle peduncle to the corpora quadrigemina (v. Bechterew).

Connections of the Superior Colliculi of the Corpora Quadrigemina

Afferent paths, i. e., to the colliculi.

1. Fibres from the optic tract through the superior brachium directly, or after interruption in the lateral geniculate body. Probably some fibres cross to the opposite colliculus through the commissure of the superior colliculi.

2. Possibly fibres from the lateral geniculate body as continuations of the paths from the occipital cortex to the lateral geniculate body. These latter run in the optic radiation, but are centrifugal.

3. With the posterior sensory tracts of the cord through the median fillet. Probably some fibres cross to the opposite colliculus by way of the commissure.

4. With the cochlear nuclei by way of the lateral fillet.

5. Fibres between the superior colliculi and the posterior longitudinal fasciculus connecting the nuclei of the third, fourth, and sixth cranial nerves with the superior colliculus. These are part of the undemonstrated fibres mentioned in the connections of the posterior longitudinal fasciculus.

6. Probably spino-tectal fibres travelling with the tecto-spinal tract, but in the reverse direction.

7. Fibres from the substantia nigra by way of the fillet (v. Bechterew[13]).

8. Fibres from the cerebellum by way of the spinal bundle of the middle peduncle through the median fillet (v. Bechterew[13]).

Note.—The fibre paths grouped under 5 and 8 are probably the afferent cerebellar paths which control the reflex conjugate movements of the eyes and the various forms of physiological nystagmus.

Efferent paths, i.e., from the superior colliculi.

1. The tecto-bulbar and tecto-spinal tracts. These fibres emerge from the ventral borders of the colliculi. The more medially situated fibres cross the raphé to form, with the corresponding fibres of the opposite side, the fountain decussation of Meynert, just ventral to the pos-

terior longitudinal fasciculus. The destinations of the fibres of these tracts are (1) the nuclei within the brain stem; (2) undetermined nuclei in the spinal cord, most probably the ventral root cells. The fibres to the spinal cord pass by way of the anterior column of the cord (fasciculus sulco-marginalis), whether directly or after interruption being undetermined. The tracts are partly crossed.

2. Some of the fibres as they emerge from the colliculi can be traced through the tegmentum, passing to the outer side of the red nucleus, piercing the median fillet and entering the substantia nigra.

3. Fibres in the commissure of the superior colliculi as axones of the cells of the colliculi.

4. Possibly fibres to the lateral geniculate body, the path being continued thence to the occipital cortex.

Connections of the Lateral Geniculate Body

Afferent paths, i.e., to the geniculate body.

1. Fibres of the outer division of the optic tract.

2. Fibres (cortifugal) from the occipital cortex by way of the optic radiation.

Efferent paths, i.e., from the geniculate body.

1. Fibres to the superior colliculus through the superior brachium. Some fibres probably reach the opposite colliculus through the commissure of the superior colliculi. The superior brachium contains also fibres from the optic tract that reach the superior colliculus without interruption in the lateral geniculate body.

2. Fibres to the occipital cortex by way of the optic radiations.

Connections of the Lateral Fillet or Lemniscus Lateralis

Afferent paths, i.e., toward the cerebrum.

1. Fibres from the superior olivary nucleus of the same side, which represent the continuation of the cochlear paths, chiefly from the opposite auditory nuclei to

the inferior colliculus and median geniculate body, and through these latter by way of the auditory radiation to the cortex. Some fibres from the superior olivary nucleus pass to the sixth nucleus, and by way of the posterior longitudinal fasciculus to the nuclei of the third and fourth cranial nerves. But see observations on the similar connections of the vestibular nucleus.

2. Fibres from the cells of the nucleus of the lateral fillet, and possibly fibres from the nucleus tegmenti lateralis of Kölliker.

3. Fibres from the acoustic striæ after mesial decussation.

The fibres of the lateral fillet end partly in the cells of the inferior colliculus, and in those of the median geniculate body, the paths being continued by axones from the cells of these bodies to the auditory cortical areas. Some fibres of the lateral fillet probably reach the inferior colliculus of the opposite side through the commissure of the inferior colliculi.

The lateral fillet mainly represents a crossed afferent cochlear path.

Connections of the Inferior Colliculi

Afferent paths, i.e., to the colliculi.

1. Fibres from the lateral fillet ending about the cells of the nucleus of the inferior colliculus of the same side, some fibres probably reaching the opposite colliculus by way of the commissure of the inferior colliculi.
2. Fibres from the cerebral cortex, especially from the temporal lobe through the inferior brachium.

Efferent paths, i.e., from the colliculi.

1. Fibres—axones of the cells of the inferior colliculi—which continue the paths of the lateral fillets interrupted in the colliculi. These fibres joining with those continued from the lateral fillet are the chief constituents of the inferior brachia.
2. Fibres to the tecto-bulbar and tecto-spinal tracts.
3. Fibres also pass by way of the superior medullary velum to the medulla of the worm.

Connections of the Median Geniculate Bodies

Afferent paths, i.e., to the geniculate bodies through the inferior brachium.

1. Fibres from the lateral fillets which end about the cells of the nuclei of the median geniculate bodies. These are the fibres that have not been interrupted in the inferior colliculi.
2. Probably fibres from the auditory cortex.

Efferent paths, i.e., from the geniculate bodies.

1. Fibres forming the lateral root of the optic tract known as the inferior commissure of Gudden. After decussation many of these fibres probably are directed toward the lenticular nucleus. Some fibres may possibly end in the subthalamic nucleus of the same side.

The existence of fibres from the median geniculate body to the median root of the optic nerve has been demonstrated. These fibres are not concerned in vision, and do not atrophy after enucleation of the eye as do retinal fibres. They probably represent in us older and little used paths for protective reflexes analogous to those paths which enable the frog, deprived of its cerebrum, to "see" sufficiently to escape objects in jumping, although it is unable to recognize anything. Similar unused paths can be traced in the olfactory organs. It is possible that these paths may still function to some extent in abnormal conditions of heightened irritability, as in the nausea of seasickness.

2. Fibres—the axones of the cells of the median geniculate bodies—which continue the paths of the lateral fillet, not interrupted in the inferior colliculi, to the auditory cortex.

Connections of the Inferior Olivary Nucleus

Afferent paths, i.e., toward the nucleus.

1. Fibres from the cerebellar cortex, the worm, and the nucleus dentatus through the restiform body, through the nucleus of the same side, mainly without interruption, across the middle line, to end in the nucleus of the oppo-

site side. These fibres constitute an efferent cerebellar path, continued, perhaps, by fibres which spring as axones of the olivary cells, and which descend in the anterior ground bundle of the cord (Kölliker). It is probable that the paths represented here cross the middle line twice. (See "Efferent paths" below.)

2. Fibres from Deiters' nucleus, which pass to the olive of the same side, thence across the middle line to the olive on the opposite side, and through the restiform body to the cerebellum. These fibres represent, in part at least, the continuation of vestibular paths interrupted in Deiters' nucleus, and are generally known as the descending vestibular olivary tract. It seems more correct to call them the crossed vestibulo-olivo-cerebellar tract for reasons stated in discussing the vestibular connections.

3. Fibres from Helweg's fasciculus, probably an afferent cerebellar pathway.

Efferent paths, that is, from the inferior olivary nucleus.

1. Fibres as axones of the olivary cells passing down in the anterior ground bundle of the cord (Kölliker). These fibres in part may represent the continuations of efferent cerebellar paths, beginning in the roof nucleus, thence to Deiters' nucleus of the opposite side, thence with or without interruption they descend to the inferior olive on the same side, and crossing to the opposite olive end about the neurones of the olivary nucleus. Such a pathway crosses the middle line twice, bringing thus each half of the cerebellum into relations with the structures on the same side of the body. See "Olivo-cerebellar fibres," under the connections of the inferior peduncle. Another possible means of double decussation of these paths is discussed under the vestibulo-spinal tract in the cord (q.v.).

2. Fibres from the inferior olive to the opposite olive, thence through the restiform body to the cerebellar cortex, the worm and the nucleus dentatus. Some fibres ascend to the cerebellum from the olive of the same side. This set of efferent olivary fibres consists mainly of afferent cerebellar fibres, and represents most probably the continuation of vestibular paths, interrupted in Deiters' nucleus, and perhaps also in either one of the olivary

nuclei. This set of fibres is mentioned under both afferent and efferent paths, because, owing to the peculiar relations of the olives to each other, the fibres that are afferent to one olive are frequently efferent in relation to the other.

The accessory olivary nuclei are two irregular, platelike masses of grey matter that lie respectively mesially and dorsally to the chief olive. Their connections are in the main those of the chief olivary nuclei.

Connections of the Superior Olivary Nucleus

Afferent paths, i.e., to the nucleus.

1. Fibres from the cochlear nuclei, mainly of the opposite side. The continuation of these paths, after interruption in the superior olive, represents the chief source of the lateral fillet. The axones of the ventral cochlear nucleus and, to some extent, those of the lateral cochlear nucleus, as they traverse the upper part of the pons toward the superior olive on the opposite side form a conspicuous transverse tract—the corpus trapezoides—that separates the tegmental from the ventral region of the pons. Some large cells found within the corpus trapezoides are known as the nucleus trapezoideus. These cells give off axones to the trapezoid body paths, and probably in part to the superior olivary nucleus.
2. Fibres from the lateral half of the cerebellum of the same side as the cerebello-superior olivary tract. According to v. Bechterew these fibres, after decussating in the middle line, pass from the cerebellar roof nucleus, and go directly or through the fibres of the trapezius to the superior olive. There is reason for believing that this is a cerebellar centripetal tract, but v. Bechterew believes it to be centrifugal because of the direct communication of the superior olive with the abducent nuclei, and because of the relation of the latter to reflex ocular movements.

Efferent paths, i.e., from the nucleus.

1. Fibres, as axones of the nucleus, which are the chief source of origin of the lateral fillet. They represent the continuation of cochlear paths to the inferior colliculus, median geniculate body, and auditory cortex.
2. Fibres to the nucleus of the sixth nerve and other

fibres by way of the posterior longitudinal fasciculus to the other oculo-motor nuclei. For reasons stated in discussing the vestibular connections with the sixth nucleus, these fibres are not to be considered as altogether mere continuations of cochlear paths, but are rather to be taken in the main as a part of many efferent paths from higher coordinating centres, e.g., the cerebellum.

v. Bechterew [13] considers these fibres as the continuation of a cerebello-superior olivary tract, carrying cerebellar centrifugal impulses, and analogous to the fibres in the superior cerebellar peduncle to the oculo-motor nucleus of the opposite side. This opinion is in harmony with the views repeatedly expressed in these pages concerning the relations between the various cranial nerve nuclei, and notably between the vestibular, sixth and third nerve nuclei.

The cerebellum consists of two lateral lobes or hemispheres connected by a median lobe, the vermis. All these divisions are subdivided into various lobules, the surfaces of which are marked by parallel transverse folds or laminæ, which give off secondary and tertiary laminæ. The general appearance presented on section is known as the arbor vitæ. The surface of the cerebellum is composed of grey matter, the cortex, enveloping the white matter. There are also masses of grey matter—the internal nuclei—within the cerebellum imbedded in the white matter. The fibres that enter the cerebellum are:

1. From the restiform body, (a) from the dorsal spino-cerebellar tract to the cortex of the vermis; (b) olivo-cerebellar fibres to the whole cortex; (c) fibres from the lateral nucleus, and possibly from other nuclei in the reticular formation.

2. The continuations of vestibular root-fibres to the vermis.

3. Fibres from the ventral spino-cerebellar tract to the vermis.

4. Fibres from the nucleus pontis to the cortex of the hemispheres.

The internal cerebellar nuclei are: (1) the dentate nucleus; (2) the roof nucleus or nucleus fastigii; (3) the nucleus emboliformis; and (4) the nucleus globosus. The globosus is connected by uncertain and limited attachments with the roof nucleus and embolus. It is also connected with the postero-inferior part of the dentate nucleus. Hence these nuclei are more or less continuous masses of grey matter. The globosus and emboliformis are but incompletely separated parts of the nucleus dentatus. The cortical cells do not send axones outside the cerebellum, all efferent paths being interrupted in the internal nuclei. The dentate nucleus receives fibres from the cortex of the hemispheres. The globosus and emboliformis receive fibres from the cortex of the vermis, whilst the nucleus fastigii receives fibres from various parts. The axones of the nucleus fastigii (fastigio-bulbar fibres), for the most part as crossed paths, pass to the vestibular and, possibly, to other reticular formation nuclei. The axones of the remaining internal cerebellar nuclei pass in the superior peduncle. There may be some efferent fibres in the middle peduncle to the reticular formation nuclei, but the greater part of this peduncle consists of pontocerebellar fibres. The inferior and middle peduncles are thus largely afferent, whilst the superior peduncle is efferent in great part to the red nucleus, thalamus, and nucleus of the third nerve.

The commissural tracts constitute part of the white matter of the cerebellum.

The anterior (superior) commissure is the larger, and lies in front of the dentate nucleus, whilst the posterior (inferior) commissure lies behind the nucleus. Each crosses the middle line to pass into the opposite hemisphere, thus constituting the anterior and posterior cerebellar decussations.

Connections of the Worm (*Vermis*)

Within the substance of the worm are:

1. The superior cerebellar commissure. This consists of fibres passing in front of the roof nucleus. Beyond the worm on either side the fibres expand into the main limbs of the medullary tree. This commissure is the main link between the cortical areas of the cerebellar hemisphere.
2. The inferior cerebellar commissure passes behind the roof nucleus as a number of small transversely coursing bundles.

3. The decussation of the roof nuclei. This differs from the commissural tract just mentioned. It consists of rounded bundles traversing the roof nucleus, especially its anterior (superior) part. More distally the fibres skirt the dorsal margin, and still farther backward they invade the beginning of the medullary limb.

4. The median sagittal bundle extends from the superior medullary velum, beneath the roof nucleus into the medulla of the worm. Above, these fibres are continued upward through the medullary velum and into the inferior colliculus.

The separate extra-cerebellar connections of the worm have not been successfully traced. Various experiments, however, point to the worm as containing most important coordinating centres with wide peripheral relations.

The superior medullary velum is a sheet of white matter extending from beneath the corpora quadrigemina to the medullary substance of the cerebellum. Laterally, it is attached to the superior cerebellar peduncles, thus forming the roof of the upper part of the fourth ventricle, where its ventral surface is lined by ependyma. Dorsally, it is overlaid by the rudimentary folia of the lingula. Its connections are:

1. Fibres of the median sagittal bundle passing between the medulla of the worm and the inferior colliculi. The function of these fibres has not been determined.

The inferior medullary velum also consists of white matter, and is attached for some distance to the front and lower surface of the nodule. Its connections, so far as known, are merely mechanical.

The fourth ventricle communicates freely with the subarachnoid space of the cord through the foramen of Magendie, situated in the median part of the roof of the fourth ventricle, and also through the foramina of Luschka in the lateral recesses of the ventricle.

Connections of the Pontine Nucleus

Afferent paths, i.e., to the nucleus.

1. Fibres from the cortex of the frontal, temporal, and occipital lobes of the same side as constituents of the fronto-cerebellar and temporo-occipito-cerebellar tracts.

2. Collaterals from the pyramidal tracts, thus establishing connections between the motor areas of the cortex and the pontine nuclei.

Efferent paths, i.e., from the nucleus.

1. Ponto-cerebellar fibres—the immediate constituents of the middle peduncle—which, for the most part, cross the middle line to reach all parts of the cortex of the cerebellar hemisphere and of the worm, and possibly the nucleus dentatus.

According to some authors the assumption that the efferent cerebello-pontine fibres end about the cells of the pontine nucleus lacks the support of more recent observations. (See note on the Connections of the middle cerebellar peduncle.)

The red nucleus consists of an ovoid reticulated field on either side of the median line in the upper half of the mid-brain, extending from the lower border of the superior colliculus to a short distance within the subthalamic region. Each nucleus consists of a complex of grey matter and fibres.

Connections of the Red Nucleus (Nucleus Ruber, Nucleus Tegmenti)

Afferent paths, i.e., to the nucleus.

1. Fibres from the superior cerebellar peduncle. As the decussation of this peduncle begins about the upper third of the inferior colliculus, and is best marked opposite the superior colliculi, it is evident that the red nucleus receives fibres as follows: (a) uncrossed fibres from the peduncle of the same side; (b) crossed fibres from the peduncle of the opposite side.

2. Fibres of the efferent cerebello-pontine tract in the middle peduncle (v. Bechterew). These fibres represent a crossed path from the half of the cerebellum on the opposite side.

3. Fibres that enter the nucleus on its lateral aspect from the cerebral cortex (Dejerine[36]) and probably also from the corpus striatum (Edinger), e.g., fibres included in the tractus strio-thalamicus, which pass from the

caudate nucleus and putamen to the thalamus, subthalamic body, and the red nucleus.

Efferent paths, i.e., from the nucleus.

1. Fibres to the optic thalamus as axones of the rubral neurones. These fibres represent the continuation of the paths of the superior cerebellar peduncle, interrupted in the nucleus. From the thalamus the path is continued to the cerebral cortex.

2. Fibres—the axones of the rubral neurones—which join the rubro-spinal tract, having crossed in the decussation of Forel. These represent the continuation of the paths from the cerebral cortex and corpus striatum, and are to be considered as indirect motor paths supplemental to the cortico-spinal pyramidal tracts.

3. Fibres—the axones of the rubral neurones—which emerge from the ventro-medial surface of the nucleus, cross the middle line in the decussation of Forel, and turn downward as the rubro-spinal tract. This latter descends within the tegmentum of the mid-brain and pons, traverses the medulla, and finally enters the lateral column of the cord.

The rubro-spinal tract, therefore, carries efferent impulses as follows:

1. From the cerebral cortex and corpus striatum of the opposite side, the path crossing once in the decussation of Forel.

2. From the cerebellum of the same side, (a) by way of the superior peduncle, the path crossing in the peduncular decussation and recrossing in Forel's decussation; (b) by way of the middle peduncle, the path crossing in the pons to reach the red nucleus and recrossing in Forel's decussation. Ferrier held that the middle peduncular fibres, after crossing the middle line, join the pyramidal tracts and recross in the decussation of the latter.

The optic thalamus is mainly a great ganglionic internode in the corticipetal paths. Most of the afferent paths from the cord, brain-stem, and cerebellum end about its cells, thence corticipetal fibres pass to all parts of the cerebral cortex and to the corpus striatum. The thalamus also receives fibres from all parts of the cerebral cortex, and from it efferent fibres proceed to the lower centres in the brain-stem and cord. The stratum zonale is a thin layer of nerve fibres on the superior surface of the thalamus. The fibres can be traced to the optic tract on the one hand and to the optic radiations on the other. The thalamus is connected with its fellow by a bridge of grey matter with few white fibres, the massa intermedia. Laterally the thalamus blends with the internal capsule. The reticulated stratum on the ventro-lateral surface constitutes the medullary lamina. It consists of numerous fibres to and from the thalamus. The ventral surface of the thalamus rests on the prolongation of the tegmental part of the cerebral peduncle, and is called the subthalamic tegmental region. The ventral nucleus receives the great sensory paths. This nucleus and the ganglion habenulæ are the oldest of the thalamic nuclei, being found in all vertebrates (Edinger[35]).

The optic thalamus, subthalamic body, and the lateral geniculate body constitute the main divisions, in regard to function, of the diencephalon. The thalamus itself contains some twenty grey nuclei (Nissl, v. Monakow). The subthalamic body and the lateral geniculate body may be regarded as grey nuclei, somewhat analogous to the nuclei of the thalamus, only more distinctly separable from the latter structure. Relatively to its bulk the region sends few fibres caudally, but it sends numerous fibres to the telencephalon (c. striatum and cortex).

Connections of the Optic Thalamus

Afferent paths, i.e., to the thalamus.

1. Fibres directly from the cord as the spino-thalamic and probably some from Gowers' tract. The relation is mainly a crossed one.

2. Fibres from the cerebellum, (a) directly, as cerebello-thalamic fibres from the same and opposite side; (b) indirectly, after interruption in the red nucleus, as the rubro-thalamic fibres.

3. Fibres from the various nuclei by way of the median fillet, i.e., (a) from the gracile and cuneate nuclei, continuing upward the paths of the posterior fasciculi of the cord after decussation for the most part; (b) from the reception nuclei of all the sensory cranial nerves, as well

as from the more extensive nuclei. (See Median fillet connections.)

4. Probably fibres of other tracts which arise within the tegmental area of the brain-stem, e.g., undemonstrated fibres from the posterior longitudinal fasciculus.

5. Fibres from all parts of the cerebral cortex, which pass in the thalamic radiation as cortifugal paths.

6. Fibres of the tractus strio-thalamicus, from the caudate nucleus and putamen. Some fibres from the caudate nucleus reach the thalamus directly by way of the internal capsule.

7. Fibres as a strand from the cortex of the olfactory bulb.

8. Fibres (thalamocipetal) from the optic tract, and from the optic radiation. These fibres constitute the stratum zonale—a layer of white matter on the superior aspect of the thalamus. The fibres from the lateral root of the optic tract are superficial, and cross the external geniculate body to spread over the thalamus. The fibres from the occipital cortex, by way of the optic radiation, invest the pulvinar.

9. Fibres of the stratum zonale from the temporal cortex *via* the ventral stalk.

10. Fibres in the mammillo-thalamic tract being a continuation of the paths leading from the olfactory cortical areas in the uncus and hippocampus to the mammillary nuclei.

Efferent paths, i.e., from the thalamus.

1. Fibres—the thalamo-cortical—which issue from the latero-ventral surface of the thalamus, and proceed to all parts of the hemisphere, some crossing to the opposite side by way of the corpus callosum. The fibres are conventionally grouped into bundles, called the stalks of the thalamus. Each stalk is named according to its relations. Thus there is a frontal, a parietal, an occipital, and a ventral stalk. The frontal stalk traverses the internal capsule between the caudate and lenticular nuclei, to which it gives fibres and ends in the frontal cortex. The parietal stalk enters the internal capsule, and frequently the lenticular nucleus in its course to the parietal cortex. Other fibres destined for the parietal lobe and

the adjacent parts of the frontal lobe, are continuations of the paths of the mesial fillet. These fibres emerge mostly from the ventral thalamic nucleus, pass outward to the under surface of the lenticular nucleus; then, bending upward, traverse the lenticular nucleus by way of the medullary striæ, or the globus pallidus to reach the cortex. Other fibres possibly continue the fillet path by entering the internal capsule, and thus, perhaps, reach the cortex directly. The occipital stalk connects the thalamus with the visual cortical areas of the occipital and parietal lobes. The fibres issue from the lateral surface of the pulvinar and as the optic radiation sweep outward and backward around the posterior horn of the lateral ventricle to reach the cortex. The ventral stalk emerges from the fore part of the ventral surface of the thalamus, arising from the lateral and mesial nuclei. It passes downward and outward beneath the lenticular nucleus, and includes two systems of fibres. Its lower part—ansa peduncularis—continues laterally into the cortex of the temporal and central lobes. Its upper part—ansa lenticularis—skirts the adjacent border of the lenticular nucleus, which it enters to gain the putamen; or, continuing through the lenticular nucleus *via* the medullary laminæ, it reaches the caudate nucleus.

The efferent fibres, i.e., from the thalamus are, therefore:

1. To the frontal cortex and to the caudate and lenticular nuclei.
2. To the parietal cortex and lenticular nucleus.
3. Fibres to the parietal and frontal lobes as continuations of the paths of the mesial fillet *via* the lenticular nucleus, the medullary striæ, or the globus pallidus to the cortex.
4. Fibres that possibly continue the fillet path to reach the cortex directly, *via* the internal capsule.
5. Fibres to the cortex of the occipital and parietal lobes.
6. Fibres of the ventral stalk, (a) to the cortex of the temporal and central lobes *via* the ansa peduncularis; (b) to the putamen as fibres of the ansa lenticularis through the lenticular nucleus, or to the caudate nucleus through the lenticular nucleus and the medullary laminæ.

7. Fibres in the thalamo-mammillary tract, the paths possibly being continued thence to the olfactory cortex by way of the anterior pillar of the fornix, or by way of the mammillo-tegmental strands to the tegmentum of the mid-brain and to lower levels. In every instance where the thalamus is connected with the cortex, i.e., where there are cortico-thalamic paths, there are also paths in the reverse direction, i.e., thalamo-cortical paths.

The epithalamus—a subdivision of the thalamencephalon—includes the trigonum habenulæ, the pineal body, and the posterior commissure.

In the trigonum habenulæ the striæ mark the site of the tænia thalami and the still deeper ganglion habenulæ. The source of the fibres of the striæ medullares is uncertain.

Probable constituents of the striæ medullares.

1. Fibres—the olfactory habenular—arising from the cells within the septum lucidum and the olfactory area.

2. Fibres—the cortico-habenular—from the cortical cells within the hippocampus and the adjacent area, and by way of the fornix and its anterior pillar, reaching the fore end of the thalamus to pass backward within the medullary striæ.

Many fibres of the striæ medullares end about the cells of the ganglion habenulæ. Some, however, reach the pineal body through the peduncle of the latter, cross in the commissura habenulæ, and end about the cells of the opposite habenular nucleus.

The ganglion habenulæ in turn gives origin to the fasciculus retroflexus of Meynert, which arches backward and downward, passing between the central grey matter of the third ventricle and the thalamus proper, then to the mesial side of the red nucleus to reach the base of the brain, where it ends about the cells of the interpeduncular ganglion. This nucleus is a well-defined collection of cells in many animals. In man it consists of a scattered median cell group within the posterior perforated space, close to the anterior border of the pons. The fasciculus, also called the habenulo-peduncular tract, receives fibres from the ganglion habenulæ of both sides, some fibres having crossed in the habenular commissure. The majority of its fibres, mostly crossed, end in the interpeduncular ganglion. Many, however, may be traced farther caudally within the tegmentum of the brain stem (Obersteiner[87]), as may also fibres from the ganglion interpedunculare.

The pineal body is situated just over the superior colliculi. Its stalk is continuous with the medullary striæ. The body contains laminated particles of carbonate and phosphate of lime. Structurally it resembles the invertebrate visual organ. It is highly developed in reptiles.

The posterior commissure (commissura posterior cerebri) provides paths by which fibres from various sources undergo median decussation. It is a small cordlike band of white matter overlying the superior entrance of the aqueduct of Sylvius. It is partially covered by the habenular commissure and the pineal peduncle above. Behind and laterally it is continuous with the superior colliculi. It is present in all vertebrates, and becomes myelinated early (Edinger[85]). The probable constituents of the posterior commissure are:

1. Fibres from the nucleus of the posterior commissure.
2. Fibres from the nucleus of the posterior longitudinal fasciculus, located in the grey matter of the third ventricle near the mammillary bodies.
3. Fibres from the posterior tract of the thalamus of the opposite side, which descend within the tegmentum, lateral and ventral to the posterior longitudinal fasciculus.
4. Fibres which cross to join the fasciculus retroflexus.
5. Fibres from the median fillet.
6. Fibres from the superior cerebellar peduncle to the thalamus on the opposite side.
7. Perhaps fibres from the deeper grey stratum of the corpora quadrigemina to the cerebral cortex of the opposite side.

The subthalamic region occupies, on each side of the middle line, a triangular area between the thalamus above and the internal capsule and its prolongation—the crusta of the peduncle—below. It is a link between the mid-brain and the diencephalon, and contains the upward prolongation of the tegmentum of the cerebral peduncles, the thalamocipetal paths of the fillet and of the superior cerebellar peduncles, the upper extremities of the substantia nigra and of the red nucleus, and a new mass of grey matter—the corpus subthalamicum.

The substantia nigra extends through the mid-brain from the upper borders of the pons, almost to the level of the mammillary body in the subthalamic region. It separates the tegmentum from the crusta of the peduncles, and contains numerous irregularly scattered nerve cells, which are pigmented. Along its ventral border lie the nuclei of origin of the third and fourth nerves, and within its lateral parts the nuclei of the mesencephalic roots of the fifth. The functions and connections of the neurones within the substantia nigra are but little known.

Probable Connections of the Substantia Nigra

Afferent paths, i.e., to the substantia nigra.

1. Fibres from the caudate nucleus and the putamen, and perhaps from the frontal cortical areas.

Efferent paths, i.e., from the substantia nigra.

1. Fibres passing into the tegmentum and the crusta, and thence to lower levels.
2. Fibres to the fillet to reach the superior colliculus (v. Bechterew[18]).

The corpus subthalamicum, or nucleus of Luys, lies just dorsal to the crusta, and lateral to the red nucleus and substantia nigra. Superiorly it extends considerably beyond the red nucleus, and consists of a network of fine medullated fibres, enclosing pigmented, multipolar nerve cells. The dorsal surface of the nucleus is defined by the overlying lateral parts of the field of Forel, which consists of a stream of fibres passing between the red nucleus and the thalamus and internal capsule.

Connections of the Corpus Subthalamicum

1. Fibres from the ventral surface of the nucleus, which pierce the adjacent crusta and join the ansa lenticularis to gain, probably, the globus pallidus.
2. Fibres—perforating—which connect the nucleus

with Meynert's and Gudden's commissures (Obersteiner[37]).

The commissura hypothalamica traverses the floor of the third ventricle above the mammillary bodies, and connects the ventro-mesial ends of the two subthalamic bodies.

The commissura hypothalamica contains also:

1. Fibres—decussating—from the anterior pillars of the fornix. These fibres reach the mammillary body as a crossed tract.
2. Fibres from the posterior longitudinal fasciculus (Edinger[35]).

The corpora mammillaria mark, by their posterior surfaces, the anterior limit of the ventral surface of the mid-brain. Each body consists of an outer layer of white matter, enclosing a core of grey substance—the nucleus mammillaris.

Connections of the Mammillary Nucleus

Afferent paths, i.e., to the nucleus.

1. Fibres from the downward arching anterior pillar of the fornix, as well as fibres through the commissura hypothalamica from the anterior pillar of the fornix of the opposite side. These fibres form part of the path connecting the cortical olfactory centres in the uncus and hippocampus with the thalamus.
2. Fibres to the mammillary nucleus—as the thalamo-mammillary tract in the mammillo-thalamic strand.
3. Possibly fibres from the posterior longitudinal fasciculus by way of the hypothalamic commissure.

Efferent paths, i.e., from the nucleus.

1. Fibres—the mammillo-thalamic tract or bundle of Vicq d'Azyr—which course upward and forward to end in the anterior nucleus of the thalamus, thus completing the path connecting the cortical olfactory centres of the uncus and hippocampus with the thalamus. Beginning in the hippocampus major this path follows the fimbria, body, and anterior pillar of the fornix to the mammillary nucleus, and thence, after interruption, proceeds *via* the mammillo-thalamic strand to the anterior nucleus of the

thalamus. This latter strand contains fibres running in both directions, between the thalamus and the mammillary body.

2. Fibres of the mammillo-tegmental tract which arch backward and downward, and are traceable into the tegmentum of the mid-brain to the vicinity of the inferior colliculli.

3. Fibres of the pedunculus corporis mammillaris. These constitute another mammillo-tegmental tract. They spring from the lateral mammillary nucleus, and course backward and downward along the medial margin of the crusta to enter the tegmentum. Their destination is not known. Kölliker believes they end in the central grey matter about the aqueduct of Sylvius, near the fourth nerve nucleus.

4. Strands from the peripheral layer of the mammillary body over the tuber cinereum (v. Lenhossék[38]).

5. Possibly fibres to the posterior longitudinal fasciculus by way of the hypothalamic commissure.

The telencephalon or end-brain consists of: (1) the hemisphærium, which includes the pallium, rhinencephalon, and corpus striatum; and (2) the pars optica hypothalami, which includes the lamina cinerea, optic commissure, tuber cinereum, and pituitary body.

The lamina cinerea consists of a thin layer of grey substance extending backward above the optic commissure, from the termination of the corpus callosum to the tuber cinereum. On either side it is continuous with the grey matter of the anterior perforated space, and forms the anterior part of the inferior boundary of the third ventricle. It connects the corpus callosum, and is sometimes called the grey root of the optic nerves (Sappe,). The functions of the lamina cinerea, if other than mechanical, are unknown.

The optic chiasm lies in the optic groove of the sphenoid bone in front of the tuber cinereum, and beneath the lamina cinerea with the anterior perforated space on either side.

The paths of optic chiasm are:

1. The visual fibres proper which, in animals below the rabbit, e.g., guinea-pig, fishes, reptiles and most birds, undergo complete decussation. In man and the

higher animals only the fibres from the inner portion of each retina cross in the chiasm to enter the opposite optic tract. The fibres from the outer side of the retina do not decussate, but pass into the optic tract of the same side.

2. Fibres that pass from one optic tract to the other along the posterior border of the chiasm (Gudden's inferior commissure). These fibres—the median root of the optic tract—connect the two internal geniculate bodies, and possibly also the inferior colliculi. They seem related more to the auditory than to the visual system, but it is possible that they are protective reflex paths.

8. Fibres that pass from the chiasm into the floor of the third ventricle, possibly to reach the third nucleus. These are the afferent paths for pupil constriction (v. Bechterew[15]), but this view seems to lack the support of evidence from the histological structure of the retina.

4. Fibres along the anterior margin of the chiasm connecting one retina or optic nerve with the other (commissura arcuata anterior of Hannover; bogen-commissure of Stilling). Lesions of one retina cause degeneration in the opposite optic nerve, due to the presence of collaterals of the optic nerve which course backward from the chiasm (Parsons[42]).

The paths in the optic tract are:

1. Fibres in the lateral root constituting the greater part, 80% in man, of the optic tract, which end in the external geniculate body, the paths being continued to the occipital cortex by new neurones passing in the thalamo-occipital radiation in company with fibres from the pulvinar and the superior colliculi (optic radiation of Gratiolet).

2. Fibres to the thalamus (pulvinar) and superior colliculi, the paths being continued to the occipital cortex by fibres that pass in the thalamo-occipital radiation. Some of the fibres from the pulvinar and external geniculate body pass as main stems or give off collaterals on their way to the occipital cortex. Of these main stems or collaterals some go to the corpus striatum, whilst others descend to the tegmentum to reach the cerebellum probably and the centres and motor nuclei of the muscles of the eyes, head and neck. These are the fibres most

probably that mediate the physiological nystagmus, which, by means of retinal impressions, facilitates the visual fixation of rapidly passing objects. It is exceedingly probable that these optic pathways are closely related functionally to the labyrinthine paths, and that in many instances they impinge with the latter on the same final common path. In the lower animals the optic lobes, which are the analogues of the corpora quadrigemina in the higher animals, are the main visual organs. Almost all the optic nerve fibres end in the mesencephalic nuclei (analogues of the superior colliculi) and in the diencephalic nuclei (external geniculate bodies). This latter is the first evidence of real visual representation in the occipital cortex (Parsons[42]). The optic nerve, in addition to the paths already mentioned, has fibres which spring from cells situated in the primary optic centres, viz., the external geniculate bodies, the pulvinar, and the superior colliculi. Though the function of these fibres is unknown, they are not necessarily centrifugal paths as Parsons suggests, but may, in part, represent afferent paths, the cell bodies of whose neurones lie in structures central to the optic tracts.

It may be observed that in fishes the vestibular nerve is in close relation with the mesencephalic centres (Loeb[43]). This shows that the mesencephalon in the lower animals at least probably contains important coordinating centres for movements of station and equilibrium.

The tuber cinereum is an outpouching, at the base of the brain, of a thin sheet of grey matter, an extension of that surrounding the cavity of the mid-brain and fourth ventricle. It contains the nucleus tuberis and the supraoptic nucleus. The connections of these nuclei are unknown.

The rhinencephalon—the oldest part of the hemisphere—includes (1) the olfactory lobe, consisting of the olfactory bulb, the olfactory tract and roots, the olfactory trigone, and the parolfactory area; (2) the uncus and a number of accessory parts. The fornix is the chief fibre tract connecting the olfactory cortex in the uncus and hippocampus with the thalamus. The olfactory cortex is, therefore, not represented in the corona radiata, but has its own special projection fibres in the cortico-mammillary tract within the fornix. In the brain the sensory paths are the first to acquire the myelin sheath, beginning with those of smell and ending with those carrying auditory impulses to the cortex. By observing the first appearance of the myelin sheath in various paths, the olfactory fibres have been traced to the uncinate gyrus, whilst the auditory and visual fibres have been traced to the temporal and occipital lobes respectively. New paths have similarly been traced from the areas in the cortex in which these sensory fibres terminate down to the medulla and motor nuclei of the cord.

Connections of the Rhinencephalon

Afferent paths, i.e., carrying impulses from the periphery.

1. To the thalamus by a strand from the cortex of the olfactory bulb to the antero-ventral part of the thalamus.
2. To the cortex (a) by the inner olfactory root, which joins the mesial aspect of the anterior extremity of the gyrus fornicatus; (b) by the outer root to the gyrus hippocampus.
3. Possibly by paths from the posterior longitudinal fasciculus, by way of the hypothalamic commissure to the mammillary body.
4. Possibly by the thalamo-mammillary pathway.

Efferent paths.

1. From the cortex by the cortico-mammillary tract from the uncus and hippocampus through the fimbria, body, and anterior pillar of the fornix to the mammillary nuclei, each mammillary body receiving fibres from the cortex of both sides, owing to a decussation in the commissura hypothalamica of some fibres from the anterior pillars of the fornix. From the mammillary nuclei the paths are continued by fibres (A) to the thalamus by the mammillo-thalamic strand or bundle of Vicq d'Azyr; and (B) to the tegmentum of the mid-brain, and possibly to lower levels, as the mammillo-tegmental tracts, of which there are: (a) The mammillo-tegmental tract proper, the fibres of which have been traced to the tegmentum of the mid-brain in the vicinity of the inferior colliculi. (b) Possibly a mammillo-tegmental tract

through the pedunculus corporis mammillaris. (c) Possibly fibres to the posterior longitudinal fasciculus from the mammillary body *via* the hypothalamic commissure. Such a connection with the posterior longitudinal fasciculus would be a possible means of bringing the olfactory centres into relation with other centres in the mid-brain, pons, and medulla. (d) Possibly strands connecting the mammillary body with the tuber cinereum.

2. From the thalamus. (a) The thalamo-mammillary tract may be an efferent path from the thalamus, along which impulses, received directly from the cortex of the olfactory bulb, may pass after interruption in the thalamus. From the mammillary body the path is continued by way of the mammillo-tegmental paths, or perhaps through the posterior longitudinal fasciculus to other centres in the mid-brain, pons, and medulla, which are thus brought into relation with the olfactory centres. (b) Possibly by fibres from the thalamus to other portions of the cerebral cortex, and perhaps to lower centres in the brain-stem and cord.

The corpus striatum is a mass of grey matter supplemental to the cortical substance. It receives fibres conveying sensory impulses, and gives off fibres, probably motor in function, which arise from its cells.

Connections of the Corpus Striatum

Afferent paths, i.e., to the corpus striatum.

1. By way of the tegmento-striate fibres, chiefly continued from the mesial fillet, and perhaps from the red nucleus and the thalamic region *via* the internal capsule, to end around the cells of the putamen and head of the caudate nucleus.

2. By the thalamo-striate fibres, which pass from the thalamus (a) by way of the internal capsule to the caudate nucleus; or (b) by way of the ansa lenticularis to the putamen; or (c) by traversing the medullary laminæ to the caudate nucleus.

8. By cortico-striate fibres. Dejerine denies the existence of these paths, but Edinger says some bundles of fibres can be demonstrated.

Efferent paths, i.e., from the corpus striatum.

1. By the strio-thalamic fibres, consisting of (a) those from the caudate nucleus to the thalamus direct; (b) those which, traversing the internal capsule and medullary laminæ and joining fibres from the putamen, pass by way of the ansa lenticularis to the thalamus; (c) those from the putamen, which reach the thalamus partly by way of the globus pallidus and partly by the ansa lenticularis.

2. By the strio-peduncular fibres, well seen in the lower animals (Edinger), as the continuation of the basal tract of the fore brain. The fibres pass from the caudate, and probably from the lenticular nucleus into the subthalamic region and the cerebral peduncle, joining in the latter the stratum intermedium, closely related to the substantia nigra.

The medullary substance of the brain consists of fibres with their supporting neuroglia. The fibres are of three kinds, viz. (1) association fibres linking the different portions of the same hemisphere together, and which, with the exception of those situated about the fissure of Rolando, are not medullated at birth; (2) commissural fibres; and (3) projection fibres.

The association fibres are divided into the short and the long. The short association fibres stretch from one convolution to another, some loops—the intracortical association fibres—being buried in the grey matter, while others—the subcortical association fibres—lie in the adjacent white matter. There are also fibres limited to convolutions of the same lobe, the intralobar association fibres. The long or interlobar association fibres include (1) the uncinate fasciculus; (2) the cingulum; (3) the superior longitudinal fasciculus; (4) the inferior longitudinal fasciculus; and (5) many other long association fibres which cannot be satisfactorily demonstrated.

The uncinate fasciculus connects the orbital surface of the frontal lobe with the anterior portion of the temporal. The inferior longitudinal fasciculus transmits visual impulses to other parts of the cortex (Dejerine).

The anterior commissure contains:

1. Fibres connecting the end of one temporal lobe with the end of its fellow of the opposite side.

2. Fibres from one olfactory lobe to the other.

3. Fibres from the olfactory lobe on one side to the hippocampal convolutions of the opposite side.

4. Fibres from the olfactory lobe through the commissure to reach the tænia semicircularis, and to proceed with it along the roof of the inferior horn of the lateral ventricle to end in the amygdaloid nucleus.

The hippocampal commissure joins the two hippocampi. These fibres cross in the psalterium, and some, after decussation, join the longitudinal fibres of the fornix and proceed to the thalamus *via* the mammillary body and mammillo-thalamic strand.

The corpus callosum—the largest commissural tract in the brain—joins the two hemispheres. Little is known definitely of the individual fibre paths or of the parts associated through them.

The projection fibres are few in the frontal, parietal, and latero-inferior part of the temporal regions. The function of these areas is not well understood. Flechsig believes them to be association centres. The olfactory cortex, as before stated, is not represented in the corona radiata, having its own projection fibres in the cortico-mammillary tract within the fornix.

The projection tracts are divided into (1) the short; and (2) the long.

The short projection tracts include:

1. The cortico-thalamic, consisting of (a) fibres from the frontal lobe to the anterior end of the thalamus; (b) fibres from the region of the fissure of Rolando, and the adjoining part of the parietal lobe to the lateral and mesial nuclei of the thalamus; (c) fibres from the occipito-temporal region to the medio-ventral part of the thalamus; (d) fibres from the posterior part of the parietal lobe, and from the occipital lobe to the pulvinar.

The thalamo-cortical tracts are associated with the foregoing. They leave the thalamus as its stalks or peduncles, and reach the various areas of the cortex. They represent continuations of the sensory paths from the cord, brain-stem and cerebellum. In these tracts are represented the median fillet, the spino-thalamic tract, and probably part of Gowers' tract and the cerebello-rubro-thalamic tract. The optic radiation represents the continuation of the visual paths of the optic tract after interruption in the pulvinar, lateral geniculate bodies and superior colliculi.

2. The cortico-geniculate and cortico-quadrigeminal tracts. These are accompanied by corticipetal fibres from the superior colliculi and lateral geniculate bodies.

3. The auditory radiation which contains cortifugal as well as corticipetal fibres running between the inferior colliculus and median geniculate body and the auditory centres in the middle portion of the superior temporal convolutions, and probably the adjoining part of the operculum. The path passes through the retro-lenticular portion of the posterior limb of the internal capsule beneath the lenticular nucleus.

4. The cortico-rubral, which is a supplementary motor tract. The origin of these fibres is probably in the cortex of the parietal lobe.

The long projection tracts include:

1. The cortico-pontine. The continuation of these paths after interruption in the pontine nucleus completes the link between the cerebral cortex and the cerebellum. The cortico-pontine tracts include (a) the fronto-pontine fibres; and (b) the temporo-occipito-pontine fibres. Through these tracts fibres from the cerebral cortex of either side reach the cerebellum on both sides.

2. The motor tracts which include:

(a) The cortico-bulbar tracts. The fibres for the movements of the eye muscles spring from the posterior portion of the middle frontal convolution (Mills[39]), adjoining the lower part of the precentral gyrus, in which are the centres for the cortico-bulbar tracts. The exact location of the strands is known only for the twelfth nerve in the posterior part of the knee, and for the seventh, which is in advance of the twelfth. Within the cerebral peduncle, the cortico-bulbar strand occupies the lateral part of the inner third of the crusta, and the fibres for the third and fourth nerves soon turn dorsally and cross the raphé to end, for the most part, in the nuclei of the opposite side. The fibres for the remaining nuclei pass near the middle line, and cross as they approach the levels of the nuclei for which they are destined.

(b) The cortico-spinal tracts or pyramids, occupy the middle third of the crusta, with the sensory paths on their outer side. They decussate for the most part at the lower boundary of the medulla. The fibres that do not decussate here are continued down the cord as the direct pyramidal tracts, but they also cross the middle line through the white commissure on reaching the level of their destination, although there is evidence tending to show that part, at least, of these fibres do not cross the middle line. (See Pyramidal tracts in the cord.)

The rolandic region is concerned in sensation as well as motion. It includes the precentral and postcentral convolutions and the paracentral lobe. The sensory fibres from the periphery to this area carry impulses which excite sensations of touch, pain, and temperature, as well as those associated with impulses from the muscles and tendons, and possibly those associated with certain phases of acts of equilibration. Excluding the sensorimotor and the various sensory areas, about two-thirds of the cerebral cortex has no known connection with the periphery. Flechsig believes these regions of the cortex to be association centres. The motor centres are mainly located in front of the central fissure. Those for the face and tongue are in the lower third of the motor zone. In the posterior parts of the second frontal convolution and in a portion of the third frontal convolution are the centres for the associated lateral movements of the eyes, and for the lateral movements of the head (Beevor and Horsley[40]). The centres for stereognostic perception and muscular sense are in the superior and inferior parietal convolutions.

The speech centres in right-handed people are in the posterior part of the third left frontal convolution, in the first left temporal convolution, and perhaps in the left angular gyrus. Broca's convolution—the third left frontal—is the motor speech centre. Destruction or disability of this centre causes motor aphasia, i.e., inability to transform concepts into words, though the patient be conscious and the tongue capable of being moved. A minor part in speech is played by the posterior part of the right third frontal convolution, but it is the chief motor centre in left-handed people.

In the first left temporal convolution is the auditory centre for speech. Lesion of this area produces loss of memory of word-sounds, though the hearing in the ordinary sense may be sufficiently good. Thus distinction is made between hearing as conceptualization and hearing in the ordinary sense of becoming conscious of sound impressions originating *ab externo*. A similar distinction holds for visual acts, and as we shall see later, there is a third manner in which individuals "see" and probably also hear and smell, and which forms the basis for protective reflex acts through the medium of centres and paths placed below, and sometimes widely separated from the cerebral cortex. The centre for memory of printed words is probably in the left angular gyrus. Lesions limited to this region are rare. They render a person unable to understand writing, though ordinary vision may be good. The existence of a motor writing centre is doubtful (Oppenheim), but if it exists it is probably located in the posterior portion of the second left frontal convolution. The centre for smell is probably somewhere near the anterior portion of the gyrus fornicatus. There is no definite knowledge about the location of the cortical centre for taste. The auditory centre is in the upper temporal convolution. Each centre is probably connected with both cochlear nerves. The frontal lobes will stand much destruction on one side without giving rise to marked physical signs. This has been frequently noted in experiments, and is a matter of common clinical experience in tumours of the frontal region, where the chief symptoms may be merely hysteroid manifestations.

The cuneus and calcarine fissure—the white line of Gennari—together constitute a primary and lower cortical or visuo-sensory centre. The lateral aspect of the occipital lobe is a visuo-psychic area, containing subareas or centres concerned in higher visual processes. Lesions of the lateral occipital lobe, especially if large and in the left hemisphere, or lesions on both sides, cause mind-blindness analogous to word deafness. Lesions of the cuneo-calcarine cortex cause lateral homonymous hemianopsia. This may also be produced by lesion of the lateral part of the occipital lobe if it extends far enough inward to interrupt the optic radiations (Schäfer and Brown[15]).

Pathways in the Spinal Cord

In a cross section of the cord the area bounded by the posterior median sulcus (dorsal septum) and the dorsal root is designated as the posterior funiculus, the area bounded by the dorsal and ventral roots is called the lateral funiculus, and that bounded by the ventral root and the anterior median fissure the anterior funiculus.

The term fasciculus is intended to cover any fairly defined bundle of fibres connecting one centre or level with another centre or level, without interruption of the pathway. A fasciculus consists therefore of but one set of neurones, e.g., the dorsal-spino-cerebellar fasciculus (direct cerebellar tract). Tract is a physiological rather than an anatomical term. It indicates a pathway for the conduction of impulses without regard to anatomical interruptions. A tract, therefore, may consist of two or more sets of communicating neurones, forming physiological continuity, e.g., the cerebello-vestibulo-spinal tract.

The term column is now reserved by many authors to designate the projections of grey matter within the white surrounding substance, e.g., the ventral grey column (ventral cornu), the dorsal grey column (the dorsal cornu).

The ascending tracts of the cord include:

1. The dorsal fasciculi (columns of Goll and Burdach).
2. The dorsal spino-cerebellar fasciculus (direct cerebellar tract, or tract of Flechsig).
3. The ventral spino-cerebellar fasciculus (Gowers' tract).
4. The spino-tectal and spino-thalamic tracts.
5. Helweg's fasciculus or bundle.

The dorsal fasciculi consist mainly of the ascending arms of the central processes of the dorso-spinal or posterior root ganglia. Cajal, in 1889, by means of the Golgi method, demonstrated the bifurcation of the posterior root fibres on entering the cord. In animals after section of the dorsal root in one of the sacral nerves, it was seen by means of the Marchi stain that a great part of the dorsal fasciculi (Burdach's column chiefly) consisted of the descending arms of the T-bifurcations of the posterior root fibres. These descending arms are destined for lower segments in the cord, and give off collaterals at various levels. Some of them constitute long pathways for spinal reflexes, e.g., the well-known scratch reflex in which clonic movements of the hind leg are evoked by scratching the region behind the shoulder of the normal dog. The paths involved in this reflex have been demonstrated by Sherrington and Laslett[46] by the method of successive degeneration, as scattered fibres in the lateral ground bundles of the cord.

The ascending arms of the T-bifurcation of the posterior nerve roots send fibres or processes and collaterals to the different segments of the cord at various levels. The remaining posterior root fibres constitute the long ascending paths of the dorsal fasciculi. These as they ascend are gradually displaced toward the middle line and away from the grey matter, in accordance with the general law of the eccentric position of the long fibre paths. Few, if any collaterals are given off from the dorsal median fasciculus (Goll's column). The zone of Lissauer consists of the bifurcation of the finer fibres of the posterior nerve root.

The terminations of the fibres and collaterals of the posterior nerve-roots are as follows:

1. Some, including part of the fibres from Lissauer's zone, end about cells in the substance of Rolando. From this point the pathway is continued by intermediate neurones to the lateral ground bundle and so to the cells of the grey matter of other segments.
2. Some pass directly through the substance of Rolando to terminate in tract cells, the axones from which pass as the spino-thalamic tract in the white matter of the same and of the opposite side. The fibres that cross the median line pass in the anterior commissure.
3. Fibres that end in relation with the ventral horn cells of the same side (reflex paths). Some reflex paths are relayed to the ventral horn cells of the opposite side.
4. Fibres that end in the tract cells of the dorsal nucleus and in other cells, the axones of which form the dorsal spino-cerebellar fasciculus (direct cerebellar tract) and the ventral spino-cerebellar fasciculus (Gowers' tract).

5. Some fibres enter the posterior aspect of the dorsal cornu, and bend upward to end in the substance of Rolando.

6. Some fibres of the dorsal root also pass in the posterior commissure to reach the cells of the grey matter of the opposite side.

The collaterals and terminals of the posterior nerve root may, therefore, terminate in any part of the grey matter. The collection of marginal cells (dorsal nucleus) situated near the mesial surface, close to the base of the dorsal cornu, extends from the cervical to the first and second lumbar segments, and is known as the column of Clarke (Clarke's vesicular column, or the nucleus of Stilling). From these cells the pathway is continued by means of neurones of the second order to the dorsal spino-cerebellar fasciculus (direct cerebellar tract) of the same side, and so to the vermis of the cerebellum. This origin of the dorsal spino-cerebellar tract has recently been disputed, but apparently upon insufficient grounds. It must be remembered, however, that the Golgi stain picks out only a few elements, and it is impossible to know what elements are omitted. The small cells in the substance of Rolando also send axones into the lateral ground bundle.

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The cells of origin of the ventral spino-cerebellar tract are not definitely known. It is known, however, that some fibres of this tract cross in the anterior commissure, and it is probable that these fibres spring from cells situated in the dorsal horn. The tract does not degenerate after section of the dorsal nerve roots (Mott). The dorsal spino-cerebellar fasciculus is, therefore, an uncrossed pathway, whilst the ventral spino-cerebellar tract is both crossed and uncrossed. Mott believes this fasciculus consists of two afferent bundles, one of which the ventral cerebellar, situated at the periphery, passes to the cerebellum in the superior peduncle, whilst the other—the crossed afferent tract of Gowers and Edinger—passes up on the outer side of the lemniscus to the corpora-quadrigemina and optic thalamus (Gordinier[41]). The spino-tectal and spino-thalamic tracts spring from cells about the base of the dorsal cornu, and pass up on the inner side of the ventral spino-cerebellar fasciculus to the tegmentum and thalamus. The tracts continue the paths of the posterior nerve root of the opposite side mainly.

The fasciculus of Helweg is seen only in the upper cervical cord. Its cells of origin and destination are unknown, although it has been assumed that the path is in relation with the inferior olive.

The dorsal fasciculi, viz., the fasciculus gracilis and the fasciculus cuneatus (columns of Goll and Burdach) are interrupted in the posterior nuclei of the medulla, the fasciculus of Goll ending in the nucleus gracilis and that of Burdach in the nucleus cuneatus. The fibres from these nuclei for the most part cross the middle line as the arcuate fibres about the level of the upper border of the pyramidal decussation in the lower part of the medulla oblongata. This sensory decussation marks the lowest limit of the fillet, the fibres of which continue the paths represented in the dorsal fasciculi. (See Connections of the fillet.)

There is question as to whether some of the component fibres of the dorsal fasciculi are directly continued to the cerebellum and mesial fillet without interruption in the posterior nuclei of the medulla. Kölliker, Solder, Hoche, and others maintain the affirmative.

The dorsal fasciculi are mainly pathways for impulses related to the so-called deep sensation (muscle and tendon). There are also in the median fibres of the dorsal fasciculi some fibres for the transmission of tactile impulses. Some authors maintain that the pathway for tactile sensation is uncrossed and interrupted in the grey matter. It is most probable, however, that the paths for tactile sensation are both crossed and uncrossed, and that in the dorsal fasciculi they travel to a variable extent, many passing all the way up to the medulla to join the mesial fillet after interruption in the posterior nuclei of the medulla, whilst others, after travelling a variable distance, pass into the grey matter.

The dorsal spino-cerebellar and the ventral spino-cerebellar paths convey those afferent unconscious impressions that underlie coordination and cerebellar muscle tonus. The dorsal spino-cerebellar path is uncrossed, whilst the ventral spino-cerebellar path is both crossed and uncrossed. Both pathways are interrupted in the cord.

The spino-tectal and spino-thalamic pathways convey impulses concerned in the sensation of pain and temperature and to some extent those of touch. These are interrupted pathways and are mainly crossed in the cord. They join the mesial fillet above the sensory decussation in the medulla.

Helweg's fasciculus probably conveys, through the inferior olive to the cerebellum, impulses akin to those that travel by way of the dorsal and ventral spino-cerebellar pathways. Some fibres of the ventral spino-cerebellar fasciculus probably reach the thalamus, and perhaps may be considered as stray fibres from other pathways, e.g., spino-tectal, spino-thalamic. (See Connections of superior and middle peduncles of the cerebellum.) Gowers believed that part of the ventral spino-cerebellar fasciculus conveys impulses related to pain and temperature. The cerebellar fibres of this tract pass by way of the inferior and superior peduncles, some fibres passing also, according to Hoche, by way of the superior medullary velum. The fasciculus has been traced as low as the fifth lumbar segment (Mott,[101] Russell[102]), while above, the tract was traced by Rossolimo[103] to the inferior colliculi, substantia nigra and the globus pallidus. The term Gowers' tract, as originally applied, included the spino-thalamic and spino-cerebellar tracts. At the present time it is restricted to the ventral spino-cerebellar fasciculus proper.

Peripheral Terminations of the Afferent Paths

The afferent or sensory paths commence at the periphery. The bodies of the afferent peripheral neurones are separated from the neural tube, and are located in the posterior spinal ganglia or their analogues in the cranial nerves. The first, second, and eighth cranial nerves have a peripheral apparatus of peculiar structure.

In the olfactory nerve the primary afferent neurone remains in the peripheral epithelium. In the eighth nerve the path originates in the neuro-epithelium, but the cell body remains permanently bipolar. The optic nerve is in reality not a nerve, being developed from an invagination of the brain wall.

The peripheral endings of the sensory neurones are:

1. Free endings, in which the nerve fibres lose their medulla and end between (not within) the epithelial cells of the skin and mucous membrane, and also between the connective-tissue strata. The finer threads often terminate in end knobs. These endings are believed to be the receptive organs for painful stimuli.

2. Fibres from the spinal ganglia, ending in the salivary glands. These fibres probably constitute part of the afferent paths for the reflex increase of salivary secretion.

3. Fibres from the Gasserian ganglion, ending in the teeth pulp.

4. Modified forms of diffuse termination, e.g., the fibres of the glosso-pharyngeal ending in clusters of cells known as the taste buds of the tongue. Taste buds represent endings of the neuro-epithelial type, having special connections with the nerves, e.g., wrappings of the nerve fibres about the taste-bud epithelia. It is believed that these accessory structures and the peculiar disposition of the nerve fibres in relation to them render the nerve fibres accessible to certain kinds and degrees of stimuli only.

5. Touch cells. In the simplest form the nerve fibres form a cuplike expansion for the reception of one end of a single cell. This form represents the beginning of the development of Meissner's corpuscles, the second stage of which is represented by the nerve fibres forming a flat disc-like expansion containing neuro-fibrils between two epithelial cells. These endings are found in the papillæ of the skin and in other parts of the body.

6. End bulbs, consisting of a granular core of cells surrounded by connective-tissue lamellæ, the nerve fibre ending in a snarl of fibrillæ twisted about the core. These endings are in the conjunctiva, glans penis, serous membranes, mesentery, etc.

7. Corpuscles of Vater-Pacini. These bodies consist of a capsule composed of crescentic lamellæ which are covered with endothelial plates. Inside the capsule is the core containing cells and naked axis cylinders. These corpuscles are always located deeply, e.g., in the deeper portions of the connective-tissue layer of the skin of the palmar and plantar aspect of the fingers and toes, in the pancreas, and in the mesentery. They are assumed to be

organs adapted for heavy pressure stimuli, whilst Meissner's corpuscles are looked upon as the organs for lighter pressure sensation.

8. In tendons the Golgi-Mazzini organs, which consist of rich arborizations of nerve fibres, forming varicosities between the tendon fibres, the whole being enclosed in a connective-tissue capsule. These organs aid in the perception of the position and movement of the limbs in space.

9. Muscle spindles which are modified muscle fibres, receiving a special nerve ending. These are widely distributed in the body and are probably present in all skeletal muscles. They are especially numerous in the small muscles of the hand and foot. None have been found in the intrinsic muscles of the tongue, nor in the eye muscles, although in the tendons of the latter Golgi-Mazzini organs have been demonstrated. Each spindle consists of a capsule composed of half a dozen concentric layers of fibrous tissue, enclosing a group of from three to ten or more muscle fibres with medullated nerves, blood-vessels, and interspersed connective tissue. The muscle fibres of the spindle are embryonic in structure, having more nuclei than the ordinary muscle fibres, and containing some undifferentiated sarcoplasm. Sherrington has shown that these structures are afferent in function, since they do not degenerate after section of the motor nerves.

10. It is still a question as to whether sensory impulses from the periphery pass into the cord by way of afferent sympathetic fibres that enter the posterior spinal ganglia.

The descending paths in the cord include:

1. The pyramidal tracts consisting of (a) the crossed or lateral cerebro-spinal fasciculus; (b) the uncrossed or mesial cerebro-spinal fasciculus.
2. The tecto-spinal tract.
3. The rubro-spinal tract.
4. The tract from the interstitial nucleus of Cajal.
5. The tract from Deiters' nucleus.
6. The tract of Thomas.
7. The septo-marginal tract.
8. The comma tract of Schultze.

The fibres of the pyramidal tracts end about the cells of the ventral horn (ventral grey column). Some observers, however, believe they terminate about the cells of the intermediate grey matter, the impulses being relayed by short neurones to the ventral horn cells. It is often stated that all the fibres of the direct pyramidal tract cross the median line at their levels of destination, but this point cannot be considered as proven, since lesion of the direct pyramidal tract causes some impairment of function in the muscles on the side of the lesion. Moreover, even in the crossed pyramidal tracts, there are some homo-lateral, i.e., uncrossed fibres.

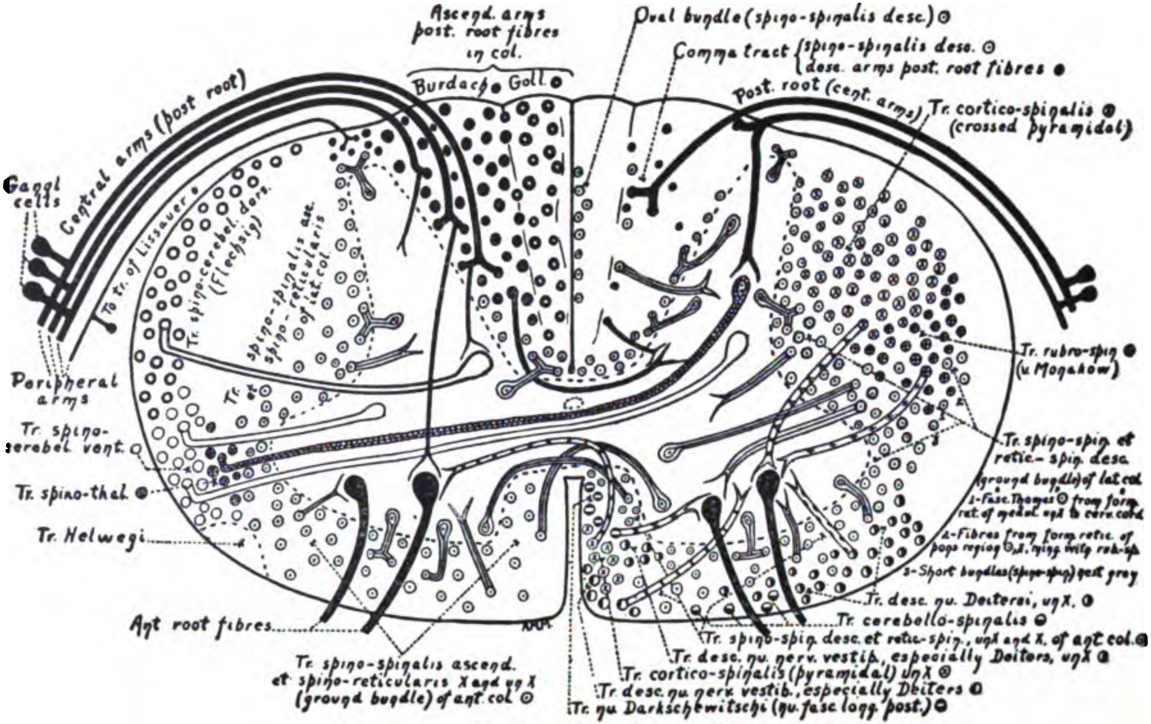
The fibres of the tecto-spinal tract spring from the roof of the mid-brain. After decussation the fibres pass downward into the cord, where they lie according to some observers near the ventral sulcus, whilst according to other observers, they are scattered through the antero-lateral ground bundle. The fibres are believed to end in relation with the ventral horn cells. The presence of any tecto-spinal fibres in the cord has been disputed.

The fibres of the rubro-spinal tract (von Monakow's tract) spring partly from cells in the red nucleus located in the tegmentum of the mid-brain, and partly from other cells in the formatio reticularis in the region of the pons. The path is mainly crossed. In the cord the tract lies ventral to the lateral pyramidal tract, its fibres partly mingling with those of the latter. The fibres of the rubro-spinal tract end in the dorsal part of the ventral horn. This tract is part of an important cerebellar efferent path, composed of three sets of neurones, viz.: (1) from the cerebellar cortex to the nucleus dentatus; (2) from the nucleus dentatus to the red nucleus; (3) from the red nucleus (rubro-spinal tract proper) to the ventral root cells. As previously stated, owing to a double decussation, viz., in the superior peduncles and in the rubral neurones of the rubro-spinal tract, efferent cerebellar impulses, transmitted *via* the cerebello-rubro-spinal tract, are mainly distributed to the ventral root cells of the homo-lateral side.

The tract from the interstitial nucleus of Cajal is uncrossed and lies near the ventral sulcus. Its fibres terminate in the ventral horn, some having been traced into the lumbar region of the cord. The interstitial nucleus of Cajal is located in the formatio reticularis of the tegmentum of the mid-brain anterior (cephalad) to the III nucleus. The fibres of this tract, however, are believed by some to originate in the nucleus of Darkschewitsch, and by others in the nucleus of the posterior longitudinal fasciculus, and by still others in the nucleus of the posterior commissure. It is a question whether any of these nuclei is identical with the interstitial nucleus of Cajal.

The tract from Deiters' nucleus occupies the ventral and mesial periphery of the cord. The more lateral fibres are uncrossed, whilst those near the ventral sulcus spring from the nuclei on both sides. It is probable that with the fibres of this tract other fibres descend from other portions of the vestibular nucleus, and from other nuclei in the formatio reticularis grisea of the medulla (reticulospinal fibres). The fibres all terminate in the ventral horn cells.

As previously stated, Deiters' nucleus is an important relay station for afferent labyrinthine impulses to the cerebellum. It is possible that Deiters' nucleus may serve as an immediate coordinating centre for labyrinthine impulses; but for reasons often reiterated in the preceding pages this is highly improbable in man and the higher animals, in whom the chief coordinating centres for labyrinthine impulses are located in the cerebellum and possibly in the mid-brain. On the other hand, Deiters' nucleus receives fibres from the cerebellum and relays the paths down the cord. Again some fibres from Deiters' nucleus, probably the continuation of vestibular paths, pass to the homo-lateral inferior olive, and directly, or after interruption, pass to the contra-lateral olive which sends numerous fibres to the cerebellum *via* the inferior peduncle (restiform body). The inferior olive and Deiters' nucleus are thus in intimate relation with the cerebellum by means of afferent and efferent paths. The afferent vestibular paths from Deiters' nucleus to the nucleus fastigii are mainly crossed. The efferent cerebellar paths from the nucleus fastigii to Deiters' nucleus are also mainly crossed, but, as many of the fibres of the vestibulo-spinal tract near the ventral sulcus are crossed, provision is thereby made for bringing each half of the


Ascend. arms post. root fibres in col.
Burdach ● Goll ⊙
Oval bundle (spino-spinalis desc.) ⊙
Comma tract {spino-spinalis desc. ⊙ desc. arms post. root fibres ●
Post. root (cent. arms)
Tr. cortico-spinalis ⊗ (crossed pyramidal)
Gangl cells
Central arms (post root)
To tr. of Lissauer
Tr. spino-cerebel. dors. (Flechsig)
spino-spinalis asc. spino-reticularis of lat. col.
Tr. o. z.
Peripheral arms
Tr. spino-cerebel. vent. ○
Tr. spino-thal. ◉
Tr. Helwegi
Ant root fibres
Tr. spino-spinalis ascend. et spino-reticularis X and un X (ground bundle) of ant. col. ⊙
Tr. rubro-spin ● (v. Monakow)
Tr. spino-spin. et retic.- spin. desc. (ground bundle) of lat. col.
1-Fasc. Thomas ⊙ from form. ret. of medul. un X to cerv. cord
2-Fibres from form. retic. of pons region ⊙ 2, ming. with rub-sp.
3-Short bundles (spino-spin.) post. gray
Tr. desc. nu. Deiterei, un X. ◐
Tr. cerebello-spinalis ⊖
Tr. spino-spin. desc. et retic-spin., un X and X, of ant col. ◉
Tr. desc. nu. nerv. vestib., especially Deitors, un X ◉
Tr. cortico-spinalis (pyramidal) un X ⊗
Tr. desc. nu. nerv. vestib., especially Deiters ◐
Tr. nu. Darkschewitschi (nu. fasc. long. post.) ⊖

(From "Histology," by Dr. Bailey. Wm. Wood & Co., Publishers) cerebellum into functional relationship with the homolateral half of the body by means of a double decussation, as in the case of the cerebello-rubro-spinal paths.

For reasons previously stated, it is perhaps better to call the vestibulo-spinal tract the cerebello-vestibulo-spinal tract and the vestibulo-olivary tract the vestibulo-olivo-cerebellar tract. (See Vestibular connections.) All the fibres of the tract from Deiters' nucleus, taken collectively, are sometimes called the antero-lateral descending tract or the marginal bundle of Löwenthal.

The tract from the interstitial nucleus of Cajal and the mesial portion of the tract from Deiters' nucleus constitute the major portion of the descending fibres of the median longitudinal fasciculus an important bundle in the segmental brain. Some observers hold that some fibres from the cerebellum pass down the cord without interruption in Deiters' nucleus.

The fibres of the tract of Thomas originate in the formatio reticularis of the medulla, and pass downward in the lateral column, to end in the grey matter of the cervical cord.

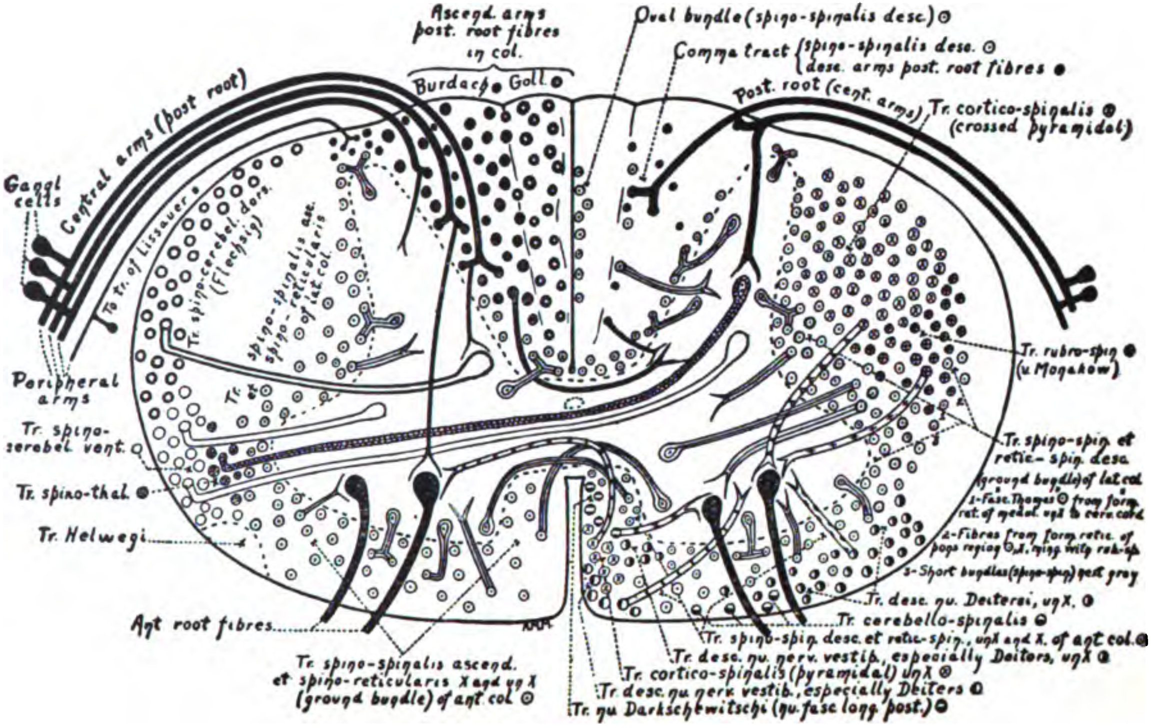
The septo-marginal tract is a small bundle of fibres lying next to the posterior septum. In the sacral cord it forms a small dorso-medial triangle, whilst in the lumbar region it forms a superficial bundle, and the oval bundle of Flechsig at the middle of the posterior septum. In the thoracic and cervical regions the fibres are scattered. The fibres of this tract are probably the descending axones of cells in the cord, forming short, intersegmental (spino-spinal) tracts.

The "comma" tract of Schultze consists of a small bundle of descending fibres, lying about the middle of the posterior column. It is a well-marked feature of the dorsal cord. The fibres probably spring from the column cells of the grey matter of the cord, forming short intersegmental (spino-spinal) tracts, although they are believed by some to be descending branches of the dorsal root fibres.

Many descending tracts contain also ascending paths.

The peripheral terminations of the efferent paths are:

1. The motor end plates of striated muscle. These consist of flattened expansions of undifferentiated sarco-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Tr. desc. nu. Deitersi, unX. ◐
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Many descending tracts contain also ascending paths.

The peripheral terminations of the efferent paths are:

1. The motor end plates of striated muscle. These consist of flattened expansions of undifferentiated sarco-

plasm. Here the nerve fibres ramify in close relation with the muscle fibre. It is a question whether the nerve terminals are located outside or inside the sarcolemma. Section of the motor nerve fibre is followed by atrophy of the muscle.

2. Axones which form the preganglionic fibres of the sympathetic nervous system. These axones always end in a sympathetic ganglion. From this point the pathways are continued by the axones of sympathetic neurones (post-ganglionic fibres of Langley), to end in the muscle of the intestines, blood-vessels, heart, etc., or in the glands, etc., of the skin. In glands the fibres end about the lumen. Some endings occur in the follicular cells of the ovary but none reach the ovum itself. In the kidney the endings are mainly in the walls of the blood-vessels. There are peripheral ganglia in the plexuses of Auerbach and Meissner, situated between the coats of the gastro-intestinal tract, mainly in the small intestine. These ganglia, which are not of the sympathetic type (Langley[50]), contain multipolar cells the dendrites of which lie in the connective-tissue layers. There are also, in the stroma of the mucous membrane, cells from which fibres pass between the epithelial cells. The spinal preganglionic fibres become pilo-motor, vaso-motor, or secretory fibres, according as their post-ganglionic continuations end in the erector muscles of the hairs, in the muscles of the blood-vessels, or in the sweat-glands, etc. It is probable that some sympathetic afferent paths reach the posterior spinal ganglia. In the heart muscle the sympathetic fibres ramify and often end in expansions.

The central nervous system may be divided into (1) a segmental part, comprising the spinal cord and the basal part of the brain; and (2) a suprasegmental part, comprising the expanded portions of the dorsal wall of the neural tube, viz., the pallium, corpora quadrigemina and cerebellum (Bailey[275]).

Tracts which connect one region of the cord or segmental brain with another region are known as intersegmental tracts, whilst tracts that pass to or from the suprasegmental parts are called suprasegmental tracts. The intersegmental tracts are mainly located in the anterolateral ground bundle close to the grey matter, some being found also in the ventral portion of the posterior columns (oval bundle, etc.).

The existence of reflex spino-spinal paths is shown by the familiar extensor-thrust reflex described by Sherrington.[47] In a spinal dog, slightly stroking the skin behind the plantar cushion with the edge of a piece of paper, or pushing the finger-tip between the plantar cushion and the toe-pads causes the leg on that side to extend powerfully for a short period. The pathway for this reflex is *via* the posterior root fibres direct to the ventral horn cells of the same side. The stimulation passes to neurones which innervate all the extensor muscles of the leg and there is but one synapsis between the afferent fibres and the body and dendrites of the efferent neurones. Reference to the schalt-zellen of v. Monakow, which are probably interposed between the afferent and efferent neurones, is omitted for the sake of simplicity.

The extensor thrust reflex is probably an important element in the reflex mechanism of locomotion. In certain cases irritation of the foot causes extensor thrust in the opposite leg. Here an intermediate spino-spinal neurone carries the stimulus to the ventral horn cells of the opposite side. In this reflex, therefore, three neurones are involved, viz.: (1) An afferent peripheral; (2) a heteromeric spino-spinal neurone; and (3) an efferent neurone

Longer spino-spinal neurones have been shown to exist by means of the scratch reflex. The pathway for this reflex is a long and uncrossed one. The fibres of the intermediate neurones actually involved, lie scattered in the lateral part of the lateral column, i.e., in the lateral ground bundle, as demonstrated by Sherrington and Laslett[45] in the following manner: The cord was transected between the second and third thoracic segments. This caused degeneration of all fibres entering the cord from the brain, mid-brain, bulb, and cervical and first and second thoracic segments. The dog was kept alive for one year, which allowed time for complete degeneration of the severed tract and for absorption of the products of degeneration. The cord was then transected again between the fourth and fifth thoracic segments and the secondary degeneration in the divided tracts studied.

by the ordinary methods, e.g., Marchi, etc. By means of this method of "successive degeneration" the scratch reflex chain was shown to consist of (1) a receptive neurone from the skin to the spinal grey matter of the corresponding spinal segment for the shoulder; (2) a long descending proprio-spinal neurone from the grey matter of the shoulder segment to that of the leg segments *via* the lateral part of the lateral column; (3) a motor neurone to a flexor muscle.

This chain has three neurones, enters the grey matter twice, and has two synapses exclusive of the schalt-zellen. The motor neurone is the final common path the rest of the arc being afferent (Sherrington[48]).

The efferent paths of the cord convey impulses which may be grouped as follows:

1. Motor, including (a) voluntary and reflex motor, muscle tonus, etc.; (b) vaso-motor; (c) viscero-motor; (d) cardio-motor; (e) pilo-motor.
2. Secretory to the various glands—gastric, salivary, pancreatic, sweat, etc.
3. Inhibitory for each of the foregoing.

The impulses conveyed in the afferent paths of the cord may be grouped as:

1. Sensory, including all impulses affecting conscious perception, e.g., visual, auditory, olfactory, gustatory pressure, pain, temperature, hunger, thirst, etc.
2. Reflex, including those impulses which evoke various motor and secretory reactions. The impulses which originate in the specialized nervous structures of the semicircular canals and vestibule of the internal ear fall within this group.
3. Inhibitory. Little is known definitely of the nature of the impulses of this group.

It has not been demonstrated that afferent impulses inhibit conscious sensations, although unconscious reflexes, e.g., sneezing may in some cases be inhibited by afferent impulses (Howell[51]).

The impulses that originate in the semicircular canals and vestibule of the internal ear can unquestionably inhibit certain efferent motor (tonus) paths, as may be seen in cold irrigations of the external auditory canal, and in certain forms of rotation. But this effect should in some instances be classed as a depression of function in the afferent elements of the ordinary reflex tonus mechanisms, rather than as an instance of positive inhibition. This subject will be discussed more fully in a later chapter.

The tonus of the muscles of the body is partly maintained by a constant stream of afferent impulses from the periphery to the related cells in the ventral horns and in other centres of coordination and tonus, e.g., the cerebellum, mesencephalic centres, etc.

The grouping of the various cells in the ventral horn has only been partially determined in regard to their functions. The ventral group is probably related to the long flexors and extensors of the limbs and the central group to the muscles for the finer movements of the fingers and toes. In the third to the fifth cervical segments the central group contains the cells related to the phrenic nerve. The dorsal mesial group seems to be related to the muscles of the vertebral column. Extra groups of cells are found in the dorso-lateral region. These cells are probably related to the muscles of the limbs.

Touch, pain and temperature are grouped as superficial sensibility, as compared with muscle and tendon sense and deep pressure, which are classed as deep sensibility.

Head and Rivers[53] have found in the skin two systems of sensory fibres. One system, the protopathic, is related to sensations of pain and of extreme changes of temperature. The sensations, however, are imperfectly localized, and the sensibility is low, i.e., the threshold of stimulation is high. The kind of sensation present in the viscera, also mediated by this system of nerve fibres, is called protopathic sensibility, and may be regarded as a defensive agency against pathological changes. It comprises sensations of pain, of heat above 37° C., and of cold not above 26° C. It is assumed that three different sets of nerve fibres mediate each of these three sensations.

The second system of fibres, the epicritic, mediates sensations from light pressures and from small differences in temperature between 26° and 37° C.

Epicritic fibres regenerate much more slowly after lesions than protopathic fibres. They enable us to make exact discriminations of touch and temperature. They are found only in the skin, and include separate fibres for heat, cold, tactual localization and tactual discrimination.

Some authors assume that the peculiar structure of the various sensory terminals (receptors) have much to do in determining the adequate stimulus for the various nerve fibres; and, indeed, there is evidence that the doctrine of specific nerve energies applies to the cutaneous senses, i.e., that each sense has its own nerve fibres capable of mediating only its own quality of sensation.

There is also evidence that the peripheral terminals of the efferent paths (effectors) determine the effect of efferent impulses. Thus Langley[53] caused fibres from the chorda tympani, which are vaso-dilators (inhibitory) for the submaxillary gland, to grow down into the peripheral end of the divided cervical sympathetic, which carries vaso-constrictors for the same gland, and found, after regeneration had taken place, that stimulation of the chorda tympani then caused vaso-constriction in the submaxillary gland. Erlanger[54] cut the fifth cervical nerve, and sutured the proximal stump to the distal stump of the sectioned vagus. Later he found that stimulation of the fifth trunk caused typical vagus phenomena.

In the following pages we shall have occasion to revert to this subject, when it will be shown that the ampullary nerve terminals of the semicircular canals and of the maculæ acusticæ of the vestibule may be affected by various forms of stimulation, e.g., galvanic, thermic, rotation and always with the same constant specific result.

Recapitulation of the Cerebellar Paths

Afferent cerebellar tracts, i.e., to the cerebellum.

Paths through inferior peduncle

1. By way of the vestibular reception nuclei (including Deiters') to the roof nucleus of the opposite side. Mainly crossed.

2. By way of the vestibulo-olivo-cerebellar tract. Mainly crossed.

3. Dorsal spino-cerebellar fasciculus (direct cerebellar, or tract of Flechsig). Mainly uncrossed.

4. The ventral spino-cerebellar fasciculus (Gowers' tract). Partly crossed.

5. Arcuate fibres from the nuclei of the posterior tracts; partly crossed; and some fibres from the arcuate nucleus, partly crossed.

6. Olivo-cerebellar fibres; mainly crossed. These fibres include the vestibulo-olivo-cerebellar tract for part of its extent. (See 2 above.)

7. Fibres from the nucleus lateralis; partly crossed.

8. Nucleo-cerebellar tract; mostly crossed. This tract includes No. 1 above, as well as the fibres formerly included under the term direct sensory cerebellar tract of Edinger.

9. Fibres from the third nucleus to the opposite half of the cerebellum (v. Bechterew).

Paths through the middle peduncle

10. Fronto- and temporo-occipito-cerebellar tracts. Mainly crossed.

11. Possibly collaterals from the pyramidal tracts—mainly crossed.

Paths through the superior peduncle

12. Some fibres of the ventral spino-cerebellar fasciculus (Gowers' tract); mainly crossed (Gordinier[41]).

13. Fibres from the red nucleus to the dentate nucleus; mainly crossed.

Through undefined paths

14. Fibres from various sources, bringing various centres of the brain-stem, medulla and cord into relation with each other under one common coordinating centre; fibres from the eyes directly or indirectly, whereby these aid in the maintenance of equilibrium through the operation of the cerebellum. The fibres from the eyes include pupillary paths (v. Bechterew[13]), but this point can hardly be considered as settled. Other fibres are supposed to bring the visceral movements under cerebellar control,

though it is probable that the cerebellum exerts but little direct control upon visceral movements. These fibres are said by some anatomists to pass in the dorsal spino-cerebellar tracts. Fibres, especially those from the various organs of special sense, convey impressions from the auditory, vestibular, visual and olfactory organs to the cerebellum.

Efferent cerebellar tracts, i.e., from the cerebellum.

By way of the inferior peduncle

1. Part of the cerebello-olivary fibres, the paths being continued into the anterior ground bundle of the cord (Kölliker). The destination of the fibres is uncertain, but most probably it is the ventral horn-cells.
2. The cerebello-vestibulo-spinal tract. Uncrossed probably because of double decussation. (See Vestibulo-spinal tract in the cord.)

Through the middle peduncle

3. The cerebello-rubro-spinal fibres. Relations mainly uncrossed because of a double decussation.
4. The cerebello-ponto-pyramidal or cerebello-pyramidal fibres. Relations uncrossed because of a double decussation.

Through the superior peduncle

5. The cerebello-tegmental tract, which includes (a) the cerebello-thalamo-cortical paths crossed and uncrossed; (b) the cerebello-rubro-thalamico-cortical paths, partly crossed; (c) the cerebello-rubro-spinal tract proper, the relations of which are mainly uncrossed because of a double decussation.
6. Fibres to the oculo-motor nuclei of the opposite side (v. Bechterew[13]). These fibres are concerned probably in the conjugate movements of the eyes.

Through undefined paths

7. Fibres to the posterior longitudinal fasciculus.
8. Fibres to the various centres in the mid-brain, pons, medulla, and cord, which insure coordinated action of ocular, skeletal and perhaps of the visceral muscles as

well as of the great centres in the medulla, e.g., the vagus, glosso-pharyngeal vaso-motor, respiratory, etc.

Marchi traced degenerating fibres from the cerebellum to all the cranial nerves, but Ferrier and Turner,[29] as well as Risien-Russell, have not been able to confirm his findings.

CHAPTER VI

THE SYMPATHETIC OR AUTONOMIC NERVOUS SYSTEM

The sympathetic or autonomic nervous system (Langley) is composed of neurones, the cell bodies of which lie in various sympathetic ganglia. The chief of these ganglia are:

1. The sympathetic chain from the superior cervical to the ganglion coccygeum.

2. The outlying ganglia, with or without names, but related to the former group as follows: (a) In the abdomen; the prevertebral ganglia, viz., the semilunar or cœliac, from which arises the cœliac plexus and the inferior mesenteric giving rise to the hypogastric nerve. These ganglia lie ventral to the sympathetic chain, but are in direct connection with it. (b) In the region of the head other ganglia of the same type are found, viz., the ciliary, spheno-palatine, otic, submaxillary, sublingual, etc. To these, perhaps, should be added the cardiac ganglia and those ganglia located in the plexuses of Meissner and Auerbach between the coats of the intestinal tube, although Langley[50] believes that the cells of the ganglia of the Meissner and Auerbach plexuses are not of the sympathetic type.

The continuity of the sympathetic with the central nervous system is effected by efferent fibres which leave the latter for the most part by way of the anterior spinal nerve roots, or their analogues in the cranial nerves. It is possible that some efferent fibres may leave the cord by way of the posterior nerve root. Such fibres, however, must suffer interruption in the dorsal root ganglia which contain some sympathetic cells, since they do not degenerate after section of the posterior nerve root. Cf. the antidromic impulses of Bayliss.[49]

The efferent paths from the central nervous system are, in every instance, interrupted once at least in the sympathetic ganglia. From the point of interruption the pathway is continued by sympathetic neurones to the peripheral tissues. The sympathetic pathway consists therefore of two or more neurones, one of which belongs to the central nervous system, constituting the preganglionic fibre. The path is completed by the post-ganglionic fibre that springs from a cell of one of the sympathetic ganglia. "The fibres from the spinal cord to the sympathetic ganglia connect certain cells of the spinal cord with the cells of the sympathetic ganglia in the same way as the fibres of the pyramidal tracts connect certain cells of the brain with the cells of the spinal cord. These spinal fibres become pilo-motor, vaso-motor, or secretory fibres, according as the fibres from the sympathetic cells, with which they are connected, end in the erector muscles of the hairs, muscles of the blood-vessels, or in the sweat-glands" (Langley[50]). The sympathetic system is also connected with the central nervous system by means of afferent pathways which pass by way of the posterior root ganglia, forming the afferent path for certain reflexes, and exceptionally, perhaps, for conscious sensations.

The sympathetic reflexes in the normal individual are carried on below the level of consciousness, but in certain disordered states, e.g., neurasthenia, cardiac palpitation, disorders of digestion, etc., afferent influences from the sympathetic system reach the level of conscious perception.

The preganglionic fibres of the sympathetic system spring from four regions, viz.: (1) From the mid-brain, emerging in the third cranial nerve, and passing to the ciliary ganglion; (2) from the bulbar region, emerging in the seventh, ninth, tenth, and eleventh cranial nerves; (3) from the thoracic nerves, viz., from the first thoracic to the fourth or fifth lumbar nerves and passing in general to the sympathetic chain, many fibres, however, passing without interruption to the abdominal ganglia; (4) from the sacral regions by way of the nervous erigens supplying the descending colon, rectum, anus and genital organs.

The connections between the anterior nerve roots and the chain of sympathetic ganglia are known as the rami communicantes. These are divided into (a) white rami, or those possessing a medullary sheath and which consist of preganglionic fibres; and (b) grey rami, which are non-medullated, or are but slightly medullated and consist of post-ganglionic fibres. In the cervical, lumbar and sacral regions, the rami are exclusively grey whilst in the thoracic and upper lumbar regions both white and grey rami are found. The fibres of the white rami (preganglionic fibres) may pass up or down the chain for some distance before ending in a sympathetic ganglion.

The grey rami represent post-ganglionic fibres returning from the sympathetic chain to join the anterior spinal nerves, which they accompany to their areas of distribution, especially the cutaneous areas, since the branches to the skin supply the sweat-glands, blood-vessels and pilo-motor muscles.

The paths which pass as post-ganglionic fibres in the grey rami to any one spinal nerve do not necessarily represent continuations of the paths that pass as preganglionic fibres in the white rami from the same nerve. In general, there is a great outflow of preganglionic fibres, including vaso-motor, secretory (sweat) and pilo-motor in the white rami from the first and second thoracic to the second, and even to the fourth, lumbar nerve. The continuations of those paths destined for the skin areas of the head, limbs, and trunk, return by the grey rami to the anterior spinal nerves and run with them to their destination.

The fibres for the blood-vessels, glands and walls of the abdominal and pelvic viscera, after entering the sympathetic chain, emerge without suffering interruption, and pass still as preganglionic fibres of the splanchnic nerves, to the cœliac ganglion, or in the branches that connect with the inferior mesenteric ganglion. From these points the paths are continued as post-ganglionic fibres.

The fibres for the glands, blood-vessels and plain muscle of the head region after entering the sympathetic chain pass upward along the cervical sympathetic to end in the superior cervical ganglion. From this point the paths are continued by postganglionic fibres, which emerge in the various plexuses that spring from this ganglion.

The course of the various preganglionic fibres is as follows:

1. Those from the third cranial nerve end in the ciliary ganglion, thence the path is continued as postganglionic fibres that pass in the short ciliary nerves to the iris and ciliary muscle.

2. The fibres that emerge by the VII and IX cranial nerves probably supply the glands and blood-vessels (vasodilator fibres) of the mucous membrane of the nose and mouth. Some of these fibres reach the fifth nerve by anastomosing branches and are distributed with it.

The preganglionic fibres of the VII and IX cranial nerves end in the ganglia of the sympathetic type of this region, viz., the spheno-palatine, otic, submaxillary, sublingual.

3. The preganglionic fibres of the X cranial nerve (representing also fibres from the X nucleus) are visceromotor for the œsophagus, stomach, small intestine, and large intestine as far as the descending colon. They also supply motor fibres for the bronchial musculature, inhibitory fibres for the heart, and secretory fibres for the gastric and pancreatic glands. It is well known that the central nervous system can inhibit and augment the general contractions of the stomach and intestines.

The ganglia in which the preganglionic fibres of the vagus end have not been definitely located, but probably these comprise the small, and for the most part unnamed ganglia distributed in and near the organs innervated.

Burton-Opitz[44] has demonstrated by means of the strohmur that the vagus contains no vaso-motor fibres for the stomach.

4. The preganglionic fibres from the sacral cord pass in the anterior roots of the second to the fourth sacral nerves. The branches from these roots unite to form the nervus erigens (pelvic nerve) which loses itself in the pelvic plexus without connecting with the sympathetic chain. The pelvic plexus is also formed in part from the hypogastric nerve arising from the inferior mesenteric ganglion. Through this pathway sympathetic fibres from the upper lumbar region enter the plexus.

The sympathetic fibres of the nervus erigens supply vaso-dilator fibres to the external genitals causing erection in the male. They also supply vaso-dilator fibres to the rectum and anus, and motor fibres to the plain muscle of the descending colon, rectum and anus. The preganglionic fibres of these pathways end in small sympathetic ganglia in the pelvic plexus or in the neighborhood of the organs supplied.

The accelerator fibres for the heart emerge from the anterior roots of the second, third and fourth thoracic spinal nerves (according to some authorities from the first and fifth thoracic, and even from the lower cervical nerve roots as well). The fibres pass by the white rami to the stellate or first thoracic ganglion, some ending in the inferior cervical ganglion, and thence by way of the annulus of Vieussens to the inferior cervical ganglion. Many branches leave the sympathetic system and vagus in this region and pass to the cardiac plexus and so to the heart. Hence in some of these branches accelerator fibres are found mixed with vagus inhibitory fibres. No accelerator fibres are found in the cervical sympathetic above the inferior cervical ganglion. The vagus contains some accelerator fibres as stimulation of that nerve after atropin causes acceleration of the heart.

The accelerator centre has not been definitely located. Stimulation of the upper cervical region causes cardiac acceleration, but this merely shows that the centre may be situated in this region or above it.

Certain nerve fibres carry afferent impulses to the vasomotor centres whereby reflex constriction or dilatation of the peripheral vessels is effected with consequent tendency to elevation or lowering of the general blood-pressure. Such fibres are known as pressor or depressor nerves respectively. In the dog the afferent depressor fibres for the heart run in the vagus, but in other animals these fibres form a separate bundle—the depressor nerve—discovered by Ludwig and Cyon in 1866. In man the depressor fibres most probably run in the vagus, and originate between the cardiac muscle fibres or in the walls of the aorta. These fibres are merely sensory, and, when stimulated, cause inhibition of the vaso-constrictor centre.

The normal mode of stimulation of the sympathetic system is reflex. This portion of the nervous system is, therefore, mainly concerned with involuntary action. Apparently the only reason that can be assigned for this fact is that the ultimate points of origin of the paths are located in regions other than the voluntary motor cortex (Howell[51]). However, as before stated, in certain abnormal conditions afferent impulses from the sympathetic system may give rise to conscious sensations. Animals, as Goltz and others have shown, live after severance of the nerve connections of the abdominal viscera with the spinal cord. After some primary disturbances the functions of the alimentary canal go on as usual, but the general vital resistance is much impaired, necessitating great care in the preparation and selection of food. This indicates that whilst the cerebro-spinal system normally exerts control over intestinal movements, and presumably over other visceral functions, the latter nevertheless can be, and perhaps normally are, mainly executed under the influence of mechanisms belonging to the diffuse as distinguished from the cerebro-spinal nervous system. Bayliss and Starling[56] concluded that peristalsis is a complicated reflex carried out through the intrinsic ganglia.

Summary of the Vaso-motor Nerves

Efferent fibres:

1. Vaso-constrictor fibres distributed chiefly to the skin and the abdominal viscera (splanchnic area). The blood-vessels of this area are all governed by the general constrictor centre in the medulla as well as by their own particular centres in the cord or elsewhere. They are normally in a state of tonic contraction.
2. Vaso-dilator fibres, distributed especially to erectile tissue, glands, the bucco-facial regions and muscles. The blood-vessels of these structures and areas are not under the control of a superior governing centre in the medulla, and the dilator fibres are not normally in a state of tonic activity.

Afferent fibres:

1. Pressor fibres which cause a rise in blood-pressure

by reflex stimulation of the vaso-constrictor centres, e.g., sensory nerves of the skin or any sensory nerve when powerfully stimulated.

2. Depressor fibres which cause vaso-dilatation and fall of blood-pressure by inhibition of the vaso-constrictor centre in the medulla oblongata, e.g., depressor fibres of the heart.

3. Depressor reflex vaso-dilator fibres which cause vaso-dilatation and fall in blood-pressure by stimulation of the related vaso-dilator centres, e.g., erectile tissue, congestion of glands in functional activity.

CHAPTER VII

THE PATHS INVOLVED IN PUPILLARY MOVEMENTS

The mechanism of pupillary constriction consists chiefly in contraction of the sphincter muscle accompanied by relaxation of the dilator and dilatation of the blood-vessels. Dilatation, on the other hand, is effected mainly by contraction of the dilator muscle, accompanied by inhibition of the sphincter and contraction of the vessels of the iris. The part played by the blood-vessels is a relatively unimportant factor, as it has been shown (Budge and Waller[192]) that dilatation can occur without variation in the blood-supply of the iris, whilst Salkowski[214] showed that actual constriction of the vessels may accompany pupillary dilatation. Langley and Anderson,[208] however, state that constriction of the arteries with contraction of their longitudinal fibres might be a possible factor in dilatation, but do not believe that vascular changes have much influence on pupillary movements.

The pupil is not directly under control of the will. Its movements originate (1) by reflex stimulation, or (2) by synkineses, i.e., the pupillary movement is associated with other voluntary or reflex movements. There are two chief reflexes and two chief synkineses, giving in all (1) the light reflex; (2) the accommodation synkinesis; (3) the sensory reflex; and (4) the cerebral or psychic synkinesis.

The optic nerve contains fibres from all parts of the retina. The pupillary fibres decussate partially in the chiasm and pass into the optic tract. Since destruction of the lateral geniculate body does not destroy the light reflex (v. Bechterew,[114] Henschen[115]), the pupillary paths evidently do not enter this body.

Flourens[66] believed the pupillary constrictor centre was in the corpora quadrigemina; but Knoll,[116] who was confirmed by v. Bechterew and by Ferrier and Turner,[118] showed that the superior colliculi might be removed without abolition of the light reflex. According to Bogroff and Flechsig[119] a tract passes from the optic tract directly into the stratum griseum centrale. v. Bechterew[117] believes the pupillary fibres leave the optic tract at the level between the corpus cinereum and the root fibres of the third nerve, near the entrance of the optic tract into the external geniculate body, and run thence to the posterior part of the third ventricle, where they end about neurones that send axones to the third nucleus of the same side. v. Bechterew[120] also believes that pupillary fibres pass in the superior peduncle between the third nucleus and the cerebellum. The paths are crossed and convey impulses in both directions.

Darkschewitsch[121] believes the pupillary fibres leave the optic tract near the external geniculate body and pass, through the thalamus to the ganglion habenulæ, being relayed thence through the posterior commissure to the nucleus bearing his name. But v. Bechterew and others showed that this nucleus is not related to the third nerve.

Bernheimer[122] thinks the pupillary fibres pass directly to the third nucleus. Bach,[123] however, could not confirm this view. It is still a question as to what the definite path of the pupillary fibres is. It seems most likely (Parsons[42]) that these fibres pass by the superior brachium into the superior colliculi, and by means of new connections the paths pass to the third nucleus of the same and opposite side. The difficulty about this is that extirpation of the colliculi does not abolish the light reflex. Parsons, however, states that the pupillary fibres end in the lateral portion of the colliculi which was not removed in the experiments of Ferrier and Turner.[118]

According to Sacki and Schmaus,[124] Shaeffer,[125] Argyll-Robertson,[126] Wolff,[127] Ruge[128] and others, pupillary constriction is caused in part by inhibition of dilatation. Parsons,[42] Anderson[118] and others maintain that pupillary constriction is solely effected by the constrictor centre. The grounds for such a view are: (1) After section of the third nerve variation in the illumination does not affect the pupil; (2) paradoxical pupil dilatation may arise and persist for one minute in strong sunlight; (8) the irregularity of the pupils consequent on sympathectomy is not diminished, but rather increased, by bright illumination of the eyes. These reasons are scarcely sufficient to eliminate dilator inhibition in the light reflex.

It is generally accepted that the centre for pupil constriction is located in the third nucleus. Marina[129] thinks the ciliary ganglion contains the centre for the light reflex. His experiments, however, are not convincing.

The efferent pupillo-constrictor path passes *via* the third nerve (branch to the inferior oblique) to the ciliary ganglion where, after interruption, the paths are continued in the short ciliary nerves to the iris.

Langley and Anderson,[132] Apolant,[133] Langendorff[144] and others have shown that the third nerve fibres entering the ciliary ganglion end about the cells of the latter as preganglionic fibres, the paths being continued through postganglionic fibres into the short ciliary nerves and so to the iris. The exact nature of the cells in the ciliary ganglion has been a matter of much investigation and controversy. Langley and Anderson[141] and Langendorff[142] have shown that the ganglion contains no spinal ganglion elements. The more recent work of Anderson[148] showed that after removal of the ciliary ganglion there was no degeneration of medullary fibres in the III, IV, V, or VI nerves. Jegerow[152] found that after section of the third nerve removal of the ciliary ganglion increased the dilatation of the pupil. And since excision of the superior cervical ganglion reduces the number of cells in the ciliary ganglion (Bumm[147]), and eserine does not control the pupil so efficiently after sympathetic ganglionectomy (Levinsohn[179]), we may conclude that some constrictor paths find their way to the ciliary ganglion through the sympathetic. (See also Onuf and Collins[207].)

Anderson[148] removed the ciliary ganglion in kittens, and after a few days found that by partially asphyxiating or killing, or by dividing the third nerve, the dilated pupil became smaller than the control. This phenomenon, known as paradoxical pupil contraction, he attributed to increased excitability of the paralyzed muscle. The phenomenon is not of as frequent occurrence as the parallel phenomenon paradoxical pupil dilatation occurring sometimes after removal of the superior cervical ganglion.

The pupillo-dilator tract begins probably in the mesencephalon near the third nucleus. Passing caudad by unknown paths it descends in the lateral tract of the cord. In the cat, dog and ape the paths leave the cord by the ventral roots of the three upper thoracic nerves, enter the rami communicantes, and pass to the first thoracic or stellate ganglion. From this point most of the fibres pass by way of the anterior limb of the annulus of Vieussens, some fibres also passing in the posterior limb, up the neck in the cervical sympathetic to the superior cervical ganglion. From the latter the tract enters the skull by the cervico-Gasserian strand, and runs independently of the carotid plexus to join the Gasserian ganglion; passing thence into the first or ophthalmic division of the fifth nerve, following the nasal branch, the tract finally leaves the latter to enter the long ciliary nerves, thus avoiding entrance into the ciliary ganglion. The long ciliary nerves enter the eye on each side of the optic nerve, and pass forward between the chorioid and sclerotic, through the ciliary body, to be distributed to the iris. In the course of this path lie the superior-cilio-spinal centre which is a hypothetical centre in the medulla near the XII nucleus, and the inferior cilio-spinal centre of Budge,[192] which lies in the cord between the sixth cervical and fourth thoracic vertebræ. Schiff,[211] however, showed that the inferior cilio-spinal was not an independent automatic centre. It may be noted that the above includes only efferent paths, or at least, it does not include afferent paths leading to the mesencephalic centre, although it is probable that the exclusion of light acts as a positive stimulus to the dilator mechanism (Howell[51]).

The existence of a pupillo-dilator muscle has been much studied and discussed. Maunoir[153] first propounded it, but no uniformity of opinion so far prevails. The difficulty seems to attach itself chiefly to the histological differentiation between the radial fibres and the blood-vessels of the iris, and to the interpretation of the nature of the cells in the posterior layer of the iris (Parsons[42]). The present state of the question seems to be that the anterior layer of the retinal pigmented epithelium acts as a dilator muscle (Grunert[177]). The cells on bleaching look like plain muscle fibres and stain in the same manner. Whatever be the state of the question from the anatomical point of view, Langley and Anderson[178] have demonstrated conclusively the existence of a dilator of the pupil.

Homolateral pupillary constriction following section of the cervical sympathetic was first pointed out by Parfour du Petit.[180] Removal of the superior cervical ganglion was found to cause wider pupillary dilatation than simple section of the nerve trunk (François-Franck[181] and others). The general effect of sympathectomy or of ganglionectomy are: miosis, narrowing of the palpebral aperture, projection of the nictitating membrane, retraction of the globe (Langendorff[183]), hyperæmia of the conjunctiva (Heese[186]), temporarily increased secretion of tears due to hyperæmia (Levisohn[179]), diminution of intra-ocular tension (Adamük,[189] Selenkowski and Rosenberg[184]), degeneration of the retinal ganglion cells and optic nerve (Lodato[182]), vascular injection of the eyeground upon the side of operation (Sinitzen[185]) and perhaps trophic disturbances.

Galvanic or faradic stimulation of the cervical sympathetic causes: homolateral pupillary dilatation with pupillary constriction on the opposite side (Schenk and Fuss[187]); change in the color of the iris because of displacement of its fibres; retraction of the nictitating membrane and widening of the palpebral fissure with protrusion (retraction in the rabbit, Heese) of the eyeball (Katschew[188]); constriction of the vessels of the conjunctiva, iris and retina (Heese[186]); increased intraocular tension (Adamük[189]) and diminished lachrymal secretion (Wolferz and Demtschenko[190]) the last effect being the result of vascular changes (Campos[191] and Levisohn[179]). Lodato found that slow irritation of the sympathetic, e.g., by implantation of a foreign body, causes dilatation of the homo-lateral pupil. This pupil reacts slightly to light, but more so to light flashed in the opposite pupil. Lodato attributed the phenomenon to retinal anæmia. Schenk and Fuss[187] confirmed Dogiel's observation that stimulation of the cervical sympathetic tended to cause constriction of the opposite pupil, but showed that it was due to the consensual reflex. As the latter is absent in rabbits the authors did not observe the phenomenon in these animals.

Under certain circumstances after sympathetic ganglionectomy, e.g., excitement, dyspnœa, anæsthesia, or death, the pupil may dilate instead of contracting (Langendorff,[183] Anderson,[193] Lewandowski,[194] and others). This phenomenon is known as paradoxical pupillo-dilatation. The cause is attributed by Anderson[193] to increased automatic excitability of the dilator contractile tissues, this latter condition being in turn attributed by Langendorff[183] to degeneration of the nerve elements.

The accommodation synkinesis is considered to be merely a constrictor effect accompanying accommodation, or rather convergence. The phenomenon is not considered to be a true reflex. Weber,[199] however, proved by means of prisms that constriction occurred in convergence without accommodation and not *vice versa*, and it is well known clinically that the accommodation phenomena are absent in paralysis of the third nerve. Moreover, even when the pupil is very small, it can be made to contract by convergence, whereas in fixation for distance in these instances the degree of dilatation corresponds exactly with that of the previous constriction. These considerations coupled with the presence of Golgi-Mazzini end-organs in the tendons of the ocular muscles tend to show that accommodation constriction resembles a true reflex mediated through varying tension in the different ocular muscles. Clinical observation shows that with one eye closed and held firmly so as to prevent convergence, efforts at accommodation cause little, if any, constriction.

The dilatation observed by Hensen and Volkers[12] on stimulation of the VI nucleus can be accounted for by inhibition of the internal rectus, coupled perhaps with active dilatation, as Sherrington[212] has shown by cortical stimulation that reciprocal innervation obtains between these muscles. Langley[213] has shown that the superior cervical ganglion sends post-ganglionic fibres to the III and VI nerves. The function of these fibres is not known. They were formerly supposed to be vaso-motor for the ocular muscles, but subsequent experiments have disproved this.

The sensory pupil reflex consists in dilatation induced by tactile, pathic and other sensory stimuli. Upon the application of any of the stimuli named, there is at first a rapid, short-lived, primary dilatation due to reflex augmentation of the dilator tone. This is followed by a second dilatation rapid in onset but slow in disappearing, and due to inhibition of constrictor tone (Anderson[193]).

The sensory reflex was first obtained on stimulation of the posterior fasciculi of the cord by Chauveau, who found that the effect was abolished by section of the cord above Budge's inferior cilio-spinal centre. Later Bernard[196] showed that stimulation of any sensory nerve evoked the reflex, and Balogh[197] that it could be evoked after removal of the superior cervical ganglion. v. Bechterew maintained that the sensory reflex was entirely due to inhibition, but Anderson[193] got well-marked dilatation from stimulation of the sciatic nerve after division of the pupillo-constrictor path. This dilatation, which was accompanied by the other phenomena associated with stimulation of the cervical sympathetic, was absent if the cervical sympathetic as well as the third nerve had been previously cut.

The cerebral synkinesis (psycho-kinesis, Parsons) is the name given to alterations of the pupil induced by psychic stimuli, e.g., fear, the thought of a bright light, etc. It is also to a great extent an association of ocular movements which themselves may be the result of sensory impulses (Parsons[42]). Haab[200] first described the phenomenon.

Pupil dilatation and constriction have been observed by Hitzig,[201] Ferrier,[8] Braunstein,[202] and many other physiologists upon stimulation of various regions of the brain. It was generally found that dilatation was more easily produced than constriction, but no strict localization could be made out. Most observers considered the effects as complications rather than direct results from local stimulation of the cortex. The dilatation is usually accompanied by the other known effects of stimulation of the cervical sympathetic. Section of this latter abolishes all these effects but the pupillary dilatation, and this it diminishes. With both sympathetics intact, full dilatation of the pupils occurs in epileptoid convulsions resulting from prolonged or frequently repeated excitation of cortical motor areas (Parsons[208]).

In Parsons' experiments upon cats the following areas of the brain on stimulation caused pupil dilatation after destruction of all possible sympathetic paths: (1) A considerable area about the crucial sulcus; (2) the mesial surface of the hemisphere in the occipital region near the crucial sulcus; (3) the anterior part of third or median convolution. Ferrier[8] got constriction and divergence from this latter area in the dog. In exceptional cases transitory constriction preceded dilatation.

In the occipital region, in the posterior part of the third or median convolutions, Ferrier got constriction, but Parsons could not confirm the observation upon the cat whilst François-Franck got temporary constriction followed by dilatation. Parsons got, upon the dog, results similar to those obtained upon the cat, and concluded that there probably exist in the frontal and occipital areas foci for constriction, but that these are masked by the dilator effects, which are more easily produced.

Section of the third nerve after previous division of the cervical sympathetic, left the pupil immobile and three-quarters dilated. Stimulation of the cortex was then without effect.

Section of the corpus callosum was without effect upon dilatation from cortical stimulation. Hence the bilateral effect was not due to stimulation acting through the opposite cortical areas, but to its effect upon the lower centres.

Stimulation of the anterior and posterior part of the corona radiata and internal capsule, i.e., fibres from the frontal and occipital areas, caused bilateral dilatation of the pupils. In one case there was constriction and convergence from stimulation of the posterior part of the internal capsule.

Sherrington,[205] after vagus and spinal transection which cut off impulses from above and those from below *via* the cervical sympathetic, found the pupils dilated as if in anger. This indicates that in pupil dilatation from stimulation of the cerebral cortex after section of the sympathetic, the mechanism consists in inhibition of the constrictor mechanism. Another theory bases the mechanism of this dilatation upon vascular changes in the iris. But all the vaso-motors for the iris run in the cervical sympathetic, and Langley and Anderson[206] have shown that vaso-motor changes whilst they may affect the pupil, do not do so to such an extent as to make them independent factors to any important degree.

It may be remarked that Onuf and Collins,[207] François-Franck and others believe that all the mydriatic paths are not confined to the cervical sympathetic.

Ferrier[8] obtained pupil constriction from the anterior and posterior limbs of the angular gyrus in the monkey, and from the third external or coronal convolution in dogs. Parsons[208] failed to confirm these observations, but François-Franck and Pitres[204] saw transitory constriction from stimulation of the former area.

In pigeons Ferrier observed intense pupil constriction on irritation of the middle of the convexity of the hemisphere. Schäfer[208] got marked constriction from stimulation of the quadrate lobule, whilst in monkeys Ferrier also got fairly constant homolateral pupil constriction from irritation of certain areas of the cerebellum.

Parsons concluded that the phenomena of pupil dilatation and contraction from irritation of the cerebral cortex are mere associations, since they do not occur in the absence of ocular movements. The movements obtained from the sensory areas, e.g., from the visual centres in the occipital cortex are due to ill-defined visual sensations leading to appropriate movement of the head and eyes. The mere fact of association with ocular movements tends to show that movements of the iris, like other ocular movements, have higher representation than that of merely spinal or analogous motor nuclei.

CHAPTER VIII

FURTHER ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS

Before approaching the study of the semicircular canals it is proper to note further a few fundamental facts concerning the anatomical and physiological relations of the cerebellum with other structures.

The following may be accepted:

1. The cerebellar hemisphere is in close anatomical and physiological relation with the cerebral hemisphere and inferior olivary body of the opposite side. In Ferrier's[57] case marked atrophy of the cerebellar hemisphere was found in a criminal lunatic with atrophy of the contra-lateral cerebral hemisphere. Starr has a similar specimen from a weak-minded child who had always been irregular in gait and balanced with difficulty. The child died when three years old from measles. There was found almost complete absence of the cerebellum on one side with marked atrophy of the fibres of the pons and inferior olive of the opposite side. Gudden[58] found on extirpation of one-half of the cerebellum ensuing atrophy of the opposite olive. Many instances of cerebellar defect have been discovered at autopsy in individuals who, in life, were considered to be fairly normal.

2. Each half of the cerebellum controls for the most part the movements of muscles on the same side of the body.

Ferrier's[8] experiments showed that stimulation of one side of the cerebellum was always accompanied by movement of the muscles on the side stimulated, and by certain characteristic movements of the eyes. This proposition seems a corollary of the first. Each cerebral hemisphere controls the muscles of the opposite side of the body, and at the same time is in intimate relation with the cerebellar hemisphere of the opposite side. Further developments will show that this relationship is the one best calculated to aid the function of equilibration.

3. The cerebellum acts wholly in a reflex manner, i.e., beneath the level of consciousness. This is generally admitted by investigators. Hence afferent and efferent paths are required. The afferent paths place the cerebellum in relation with the specialized sensory organs of equilibration, the chief of which are the semicircular canals, the eyes, and those structures in which kinæsthetic impulses originate, viz., the skin, muscles, tendons, articular surfaces, etc. The cerebrum, however, besides being capable of exerting control over the muscles involved in equilibration and in ocular movements has, in man, to be reckoned as an important source of afferent cerebellar impressions in acts of equilibration. The phenomena of conjugate deviation observed by Schäfer and Mott,[59] and by Risien-Russell[60] on stimulation and ablation of the ocular centres in the frontal lobe and the similar phenomena that sometimes accompany cerebral hemorrhage, closely resemble those observed on stimulation (Ferrier[8]), or ablation (Luciani[61]), of parts of the cerebellum or section of the cerebellar peduncles, or of the auditory nerve. These phenomena are best explained by assuming the existence of cerebral and cerebellar tonus balanced mechanisms, working in harmony and in a reflex manner, the cerebral motor mechanisms of one side acting in harmony with the corresponding mechanisms of the opposite half of the cerebellum.

In addition to the direct tonus influence which the cerebral centres exert upon the ocular and other muscles it would seem that the cerebrum is an important source of afferent cerebellar impressions whereby it influences the actions of the cerebellar mechanisms upon the ocular and other muscles.

In other words, afferent cerebellar paths conduct impulses from the various parts of the cerebral cortex to the cerebellum. Such cerebro-cerebellar paths are somewhat analogous to those from the semicircular canals, and from the peripheral kinæsthetic end organs. The impulses conveyed by them act probably after the manner of those conveyed by the vestibular or kinæsthetic paths in influencing cerebellar action.

In a large cerebral hemorrhage confined to one hemisphere not only is the independent action of the cerebral centres for the head and eyes suspended, but the cerebral influence exerted through the cerebro-cerebellar paths is in abeyance. The consequent conjugate deviation results from removal of the direct cerebral tonus, as well as of the associated, indirect, cerebro-cerebellar tonus on one side of the body, leaving the opposing mechanisms of the opposite side in free and unopposed control. The phenomena following section of the superior and middle peduncles of the cerebellum seem to afford grounds for this assumption. It may, therefore, be concluded that the cerebrum besides exerting tonic influence directly on muscle, can also affect muscle tone indirectly through the medium of the cerebellum. The anatomical relations strongly corroborate this view. The fact that removal of the cerebral hemispheres of the frog does not disturb the functions of equilibration to any extent, does not contravene this view because the hemispheres are relatively unimportant structures in the frog, as compared with man, and bilateral lesions tend to counterbalance each other, leaving the remaining elements of the balanced mechanisms to control acts of equilibration. The turning of the eyes to the side of the lesion in cerebral apoplexy accords with Ferrier's[8] experiments, in which stimulation of the cerebellum on one side was invariably followed by turning of the eyes toward that side. These matters shall receive further discussion in a later chapter.

The cerebellum by means of its afferent and efferent peripheral relations constantly holds the muscles involved in equilibration in a state of tone ever ready to respond to impulses originating from change of position (Ewald) or other stimulus. This tonic influence is bilateral, each half of the cerebellum controlling in the main the muscles on the homolateral side. Thus delicate cerebellar mechanisms are constantly active in the reflex maintenance of equilibrium. Sometimes these mechanisms work unaided and at other times in conjunction with the higher voluntary motor centres. Serious interference with the afferent, efferent, or central elements of the reflex arcs of these mechanisms results in disturbances of equilibrium, incoordinated action of the muscles of dynamic equilibrium and other phenomena. Such disturbances, if of mild degree, may be corrected by voluntary effort, but this latter is always awkward, very exhausting, and requires the constant attention of the individual, which is the opposite of what occurs in normal equilibration. Compare the results observed by Weir-Mitchell[62] in pigeons after injury to the cerebellum

4. The peripheral end organs of the vestibule and semicircular canals are mainly in crossed relation with the cerebellum through the vestibular nerve. v. Bechterew maintains the contradictory of this proposition. The succeeding chapters, as well as careful study of the fibre-paths, will reveal this crossed relationship. Ferrier[8] has shown that the cochlear apparatus is in relation mainly with the temporo-sphenoidal lobe of the opposite side. He found on stimulation of the auditory area in monkeys, cats, dogs and jackals, pricking of the opposite ear, wide opening of the eyes, dilatation of the pupils, and turning of the head and eye to the opposite side, thus indicating the direction of the source of the imagined sound. These experiments were repeated many times, and on both sides of the brain.

Before proceeding further, it is proper to call attention to such terms as "rolling on the long axis to the side of operation" in animals, and to contrast it with "revolving to the right or left, or to the side of the lesion, on the long axis" in man. Owing to the difference in posture normally assumed by man and the quadrupeds, these expressions have altogether a different meaning. In the case of a dog "rolling on the long axis toward the side of operation," means that the direction of the motion would be represented by a bent arrow passing transversely over the dorsal aspect of the animal, whilst in the case of man the direction would be indicated by a bent arrow passing transversely across the ventral aspect of the body. The rotations in the two instances, though in the same direction, would be, according to current forms of expression, absolutely in opposite directions. A similar difficulty is encountered in describing movements of the eyeballs occurring in nystagmus and ocular deviations. Thus, it is said, that the eyeballs rotate inward or outward, or from left to right on the horizontal antero-posterior axis. Such statements afford opportunity for misinterpretation. Thus if the upper part of the visible portion of the eyeballs be held in mind as the point of observation, the direction will be indicated by an arrow pointing one way, whilst if the lower portion of the eyeball be taken as the point of observation, the direction will be indicated by an arrow pointing in the opposite direction. Hence it seems better to drop the terms rotating inward and outward, and substitute for them rotation in the direction of the hands of a watch or the reverse, the watch in every instance being considered as held facing the observer, and in a plane parallel to the vertical transverse mesial (coronal) plane of the subject's body. Similarly in rotations with the subject in the recumbent posture the watch is considered as placed on the subject's breast with the dial looking toward the observer, and parallel to the coronal plane of the subject's body. The direction of certain forms of rotation can then be designated as with or against the hands of the watch, whilst the nystagmus, ocular deviations and subjective sensations of movement may be recorded in similar terms.

The simple reflex arc comprises, according to Sherrington, (1) a peripheral receptive portion (receptor), (2) a peripheral motor portion (effector), and (8) a conducting part which consists of the afferent and efferent neurones. "At the commencement of every reflex arc the receptive neurone is the sole avenue for impulses generated at its receptive point. The path is therefore exclusive, and other receptive points cannot employ it. A single receptive point may play reflexly on a number of different effector organs, e.g., on many muscles and glands in different regions, yet all its reflex arcs spring from one single shank, i.e., from one afferent neurone which conducts from the receptive point at the periphery into the central nervous organ. At the end of every reflex arc is the motor neurone, the last conductive link to an effector organ. This receives impulses from many receptive sources in various regions of the body. It is the sole path by which all impulses, no matter whence they come, reach the muscle fibres. The receptor neurone forms a private path exclusively, serving impulses of one source only. The effector neurone is a public or common path for impulses arising at many sources of reception. A receptive field, e.g., an area of skin is analyzable into receptive points. An effector organ stands in reflex connection not only with many individual receptive points, but with many receptive fields. Impulses generated in manifold sense organs can pour their influence into one and the same muscle. Therefore, in reflex arcs the initial neurone of each is a private path, exclusive for a receptor point or group of points, and finally the arcs embouch into a path leading to an effector organ, and their final common path is common to all receptive points wheresoever they may lie in the body, so long as they have connection with the effector organ in question.

"But arcs converge to some degree before finally converging upon the motor neurone. Their private paths embouch upon internuncial paths common in various degree to groups of private paths. The terminal path is the final common path to distinguish it from internuncial common paths. The motor nerve to muscle is a collection of (parts of) final common paths. Internuncial paths conduct and converge to final paths or to further internuncial paths. In the scratch reflex the long descending proprio-spinal neurone is connected with a whole group of afferent neurones—private paths from the scalptor receptors in the skin field of the scratch reflex. Again, in the retina and olfactory bulb, Cajal and others have shown that the conducting fibres of whole groups of receptors impinge upon individual neurones of the next relay. The thalamic neurones form a path upon which the dorsal columns, fillet, and spino-cerebellar peduncular paths converge. Therefore, each internuncial path is, to some extent, a common path, just as the receptive neurone is common to a small number of receptors. The ultimate path, therefore, differs from the internuncial path only in that it exhibits communism in the highest degree" (Sherrington).

Because each instance of convergence of two or more afferent neurones upon a third which, in regard to them is efferent, affords, as shown, an opportunity for coalition or interference of their action, each structure at which it occurs is a mechanism for co-ordination (Sherrington[64]). In simple reflexes using only one muscle it may be accepted that the motor neurone is the final common path. In complex co-ordinated reflexes involving the simultaneous action of several muscles, e.g., the reflex acts of equilibration, mediated by cerebellar neurones, the spinal motor neurones cannot be the final common path which in this instance must of necessity consist of efferent cerebellar neurones, using, however, various spinal motor neurones to manifest its influence upon the effector organs brought into play.

The receptor organs are divided (Sherrington) into three groups, as follows:

1. The exteroceptive field endowed with numerous receptive organs adapted to mechanical contact—cold, warmth, light, sound, injury (noxa); in fact, every mode of stimulation whereby the environment can affect the external surface of the body.
2. The proprio-receptive field with specific receptor organs adapted to modes of stimulation obtaining in the muscles, tendons, joints, walls of the blood-vessels, etc.
3. The interoceptive field, co-extensive with the internal surface of the body (alimentary canal, etc.), and furnished scantily with receptor organs as compared with the exteroceptive field, though these are peculiarly adapted to chemical agencies.

Receptors that respond to stimuli originating from an object at a distance from the body are known as projicient or distance receptors, e.g., those of vision, hearing, and smell. Such receptors tend to have a large cortical representation and to control the skeletal musculature as a whole.

Receptors that respond to the action of noxious agents which threaten immediate harm to the skin are known as nociceptors. Furthermore, the reflex which these receptors excite is prepotent, protects by escape or defence, is imperative and is accompanied by pain.

There is no such thing as a purely simple reflex, because the nervous system is never at rest, and no part of it is disconnected from the rest. Reflexes are more easily elicited from the skin (receptor organs) than from the afferent nerve trunk, and some are only elicited from a particular surface by particular stimuli. One reflex may combine harmoniously with another, so that their reactions mutually reinforce each other. Such reflexes are allied and their neural arcs are called allied arcs. On the other hand, some reflexes are incompatible and antagonistic, i.e., one inhibits the other or a whole group of others. The reflex or group that inhibits its opponents is called prepotent for the time being. Nociceptive reflexes override (inhibit) all others and are therefore prepotent. The type reflex, e.g., the scratch reflex, etc., results from the harmonious relation between allied reflexes and allied arcs. In a type reflex the whole motor centre potentially belongs to all and each of the groups of receptive organs proper to the reflex. The elements of the centre of the type reflex are combined and incapable of isolated excitation.

In the decerberate dog a reflex that is accompanied by certain mimetic movements simulating certain affective states, e.g., anger, pain, etc., is called a pseudo-affective reflex. Pain is the psychic adjunct of a protective reflex. The reflex is always purposive.

A receptive field frequently contains receptors of two different species, e.g., tangoceptive and nociceptive which may not both of them initiate reflexes belonging to the same type, i.e., related between themselves as allied reflexes. On simultaneous stimulation of these two kinds of receptors the nociceptive suppresses the tangoceptive reflex. This is known as reflex complication.

''The compounding of reflexes is a main problem in co-ordination. Hence the common path is a feature'' (Sherrington[64]).

''In the scratch reflex there is an end effect of positive sign followed by an inhibitory phase which is an end effect of negative sign. This succession in the reflex is repeated many times, the stimulus being continued. The scratch reflex is therefore of double sign, i.e., it develops first an excitatory and then an inhibitory end effect.

''In the flexion reflex of the hind limb of a spinal dog or cat, the end effect is expressed by two groups of muscles whose contractions act in opposed direction at the same joints. This opposition is obviated at the end of the reflex by the end effect having the form of excitatory state as regards the motor nerve to the flexor muscles, but suppression or withholding of excitatory state (central inhibition) as regards the motor neurones of the extensor. This is a reflex of double sign, whilst the scratch and eyelid reflexes are of successive double sign'' (Sherrington[63]).

Although the intimate nature of inhibition is but little understood it has been clearly demonstrated by Sherrington[65] that inhibition is an active process and an essential part of the reflex. By means of inhibition the motor neurones are precluded from the arc of one reflex whilst left open to another. In any type reflex, inhibition of certain muscles appears as the negative aspect of positive excitation in other muscles. This phenomenon which seems to be an essential part of every reflex movement constitutes what is known as reciprocal innervation. The seat of inhibition seems to be, not in the afferent or efferent neurone, but in an internuncial mechanism between them, *viz.*, the ultimate synapse. The objection made to the spinal motor neurone as constituting the final common path for complex co-ordinated reflexes of equilibrium seems to indicate that in these and similar reflexes the seat of inhibition is not placed at, or near the commencement of the spinal-motor neurone, but higher up, *viz.*, at the commencement of the cerebellar or other neurone which, in this instance, is the commencement of the final common path. Tonic reflexes of posture are the most readily inhibited.

From the foregoing it is apparent that the most intimate functional relations obtain between the various parts of the nervous system. In the preceding chapters an attempt was made to trace anatomically these connections, the study of which may help toward an understanding of the physiological relations, though it must be understood that the tracts traced only crudely and indefinitely represent the actual paths employed in the various activities of nervous system. Thus, whilst it is not possible to trace the minute anatomical connections it seems that every final motor neurone, or, at least, every common path is, to some extent, in relation with the receptive areas of all parts of the body through afferent arcs of greater or less resistance.

PART II

PHYSIOLOGY OF THE SEMICIRCULAR CANALS

CHAPTER IX

PHYSIOLOGY OF THE SEMICIRCULAR CANALS FROM THE STANDPOINT OF ANIMAL EXPERIMENTATION

So much investigation has been devoted to the study of the semicircular canals that it would take more space than present circumstances permit to give even a cursory review of the work done. As early as 1693 Bannister, an Englishman whose researches were perhaps the first experimental effort in this direction, observed the rôle of balancers in certain kinds of flies, and noted disturbances of equilibrium after their removal. Since Bannister's time the literature of the semicircular canals teems with names familiar to every student of medicine. Only a few can be mentioned, such as Scarpa, Darwin, Jackson, Flourens, Goltz, De Cyon, Högyes, Hitzig, Spamer, Brown-Séquard, Lucæ, v. Bechterew, Vulpian, Baginsky, Breuer, Mach, Crum-Brown, Laborde, Koenig, Lee, Sewall, James, Kreidl, Loeb, Biehl, Dreyfus, Schwartze, Wanner, Ewald, von Stein, Delage, Engelmann, Koryani, Steiner, Bruck, Bárány, Neumann and hosts of others.

Flourens was the first to point out the intimate relationship between the semicircular canals and the function of equilibration. He showed that injury of the membranous canals was followed by disturbances of equilibrium—varying with the seat of the lesion. De Cyon, Goltz and others confirmed Flourens's observations.

Flourens[66] and De Cyon,[67] experimenting on the semicircular canals of pigeons, obtained the following results:

Division of the horizontal canal on one side caused a series of oscillations of the head in the horizontal plane on a vertical axis. These cease in a short time, but on section of the corresponding canal on the other side they reappear with greater intensity. The bird cannot now maintain its equilibrium, but falls or turns on a vertical axis or circles round and round. After eight to ten days the bird recovers and seems normal but for a certain awkwardness seen especially in flight.

Division of the posterior vertical canals causes similar but more violent disturbances of equilibrium. The movements of the head are in a vertical plane on a horizontal axis. The pigeon somersaults head over heels. The disturbances subside in fourteen days, but a certain brusquerie of movement remains, with an almost complete inability to fly.

Division of the superior vertical canals causes movements of the head from behind forward and from right to left, or vice versa, with profound disturbances of equilibrium. The bird constantly tends to somersault heels over head. The plane of the movements of the head is diagonally around a horizontal axis. The movements on section of the canals seem therefore to take place in the plane of the canals operated on.

In rabbits, section of the canals gave results similar to those obtained in pigeons, but more enduring and the oscillations affected the eyeballs more than the head and trunk (De Cyon [67]). Section of one horizontal canal caused tendency to movements of manège (circus movements). Section of one vertical canal caused the animal to turn on the longitudinal axis. There was deviation of the eyeballs and nystagmus, the plane of the oscillations varying with the canal injured, but the oscillations of the eyeballs were more or less independent of the movements of the head.

In frogs, section of the horizontal canals caused the head to turn on the long axis of the body, the animal falling to one side or leaping. Section of the posterior vertical canals caused the animal to fall on its back. Section of the other vertical canals caused complete somersaults and the utmost disorder of movement.

On section of the superior vertical canals the frog swims in an upright position, pivoting round and round. Recovery may follow destruction of all the canals on one side. At first there is a tendency to fall to the injured side, and the leg of this side gives way as if broken. In many animals the head assumes an unnatural position, e.g., in pigeons the occiput going to the side of injury and the beak to the opposite side.

After destruction of the canals on both sides equilibration is extremely affected. Pigeons after a time learn to walk, but the disturbances of equilibration recur if a hood be thrown over the eyes. After some months the birds begin to look normal, but they cannot fly, and when suddenly startled their confusion of movement returns and they tumble about helplessly. Animals with their semicircular canals destroyed can hear (Flourens), while those in which the cochlea alone has been destroyed cannot hear but can equilibrate. This accords with what is now known and accepted, viz., the complete anatomical and physiological distinction between the vestibular and auditory fibres and nuclei of reception of the eighth nerve.

Section of the auditory nerve in frogs gave results similar to those obtained on section of the semicircular canals (Goltz [68]). Similar effects were observed in dogs by v. Bechterew [69] on section of the eighth nerve, and in sheep and horses by Biehl.[70] The animals rolled round toward the side of operation and showed a skew deviation of the eyes, that on the side of operation looking downward and outward, whilst that on the other side looked upward and inward. The oscillations of the eyes were in the direction opposite to that of the deviation. Rolling movements were most marked in the first few days, being almost incessant. When not rolling on its axis (longitudinal) the animal lies on the side of section with this side of the head downward. The legs on the side of section are doubled up close to the trunk, but flaccid, whilst those of the opposite side are rigidly extended outward. If the animal is placed in any other position than on its side, all the stiffness of the limbs ceases to be manifest (v. Bechterew [69]).

The disturbances of equilibration gradually become less pronounced, but for many weeks the animal is unsteady. This unsteadiness is greatly increased by covering the eyes. A loud sound often causes the animal to fall on the side of section or to roll round once or twice. When both auditory nerves are cut the animal can neither stand nor walk. There is no paralysis of the limbs, but all movements of them are irregular and purposeless. The head and eyes oscillate, but the eyes oscillate in a horizontal plane and there is no skew deviation as when one nerve is divided.

Various theories have been put forward to explain the modus operandi of the semicircular canal apparatus. Thus actual currents of endolymph were at first believed to be the adequate stimulus of the ampullary nerve endings (Goltz [68]); then, because the membranous canals are of capillary calibre this theory was displaced by that of partial pressures (Mach,[71] Breuer [72] and Crum-Brown [73]) of the endolymph in a certain direction that is to or from the ampulla. De Cyon [74] differs from Crum-Brown, Mach and Breuer and considers the semicircular canals as a system of physiological coördinates to which we refer all our notions of space. Breuer[75] and Ewald[76] showed that determination of an endolymph current toward the ampulla produced deviations and displacements of the head in a certain direction, while determination of an endolymph current from the ampulla toward the canal produced similar displacements, but in the opposite direction. The existence of bilateral labyrinthine tonus mechanisms which, in the erect position, constantly supply the muscles involved in equilibration with tonus impulses, makes the hypothesis of partial pressures exerted through the endolymph more acceptable without necessarily implying any gross actual movement of the fluid. Moreover, the delicacy of these mechanisms on either side of the body, each of which so nicely adjusts itself in balancing its fellow of the opposite side under varying conditions, implies rapid alterations such as could be effected by means of pressure rapidly transmitted through comparatively incompressible liquids rather than by means of the clumsy, slow movements of the liquid itself. It is not probable that in rotations the pressure exerted through the endolymph in one direction with reference to the ampulla upon one side of the body is reinforced by a pressure exerted in the opposite direction in the corresponding canal upon the other side.

This phase of negative stimulation will therefore be omitted in the discussions that follow.

The otoliths, which in mammifera are two, and in other vertebrates three on either side, are, according to Breuer [77] the peripheral organs of specific sensations of position and of movements of translation. They are disposed in two planes (three in animals possessing the lagena) perpendicular to each other, the physiological stimulus being the gravitation.

For the sake of simplicity and to avoid confusion, little mention of these organs will be made in the discussions that follow, though undoubtedly they are affected in various forms of rotations and movements as well as in prolonged aural irrigations and in strong galvanic stimulation.

CHAPTER X

THE EFFECTS OF PASSIVE ROTATION

Passive rotations were performed in various postures and in various planes. For rotation about the long axis of the body, the subject was placed in an arm-chair suspended from the ceiling. For rotation about the other axes of the body, a broad board, long enough to permit the subject to lie in any position, was suspended after the manner of a boatswain's chair.

Passive rotation in general caused various disturbances depending upon the duration (repetition) and rapidity of the rotation; upon the axis of the body about which the rotation took place; but, above all, upon the abrupt reversal, retardation or acceleration of the movement. Mild rotations repeated a few times caused merely a transitory dizziness. If repeated sufficiently often with reversals, accelerations and retardations, rotations of mild degree, i.e., of short range and of a low rate of speed, gradually produced profound disturbances in the organism.

Rotations more severe in grade caused disturbances of equilibrium with vertigo; displacements and deviations of the eyes, head and body; disturbances of the circulation and respiration; disturbances of the digestive apparatus with increased flow of saliva, nausea, wretchedness and vomiting; and finally, disturbances of the nervous system varying from a mild degree of irritability to the most profound degree of general physical and mental prostration. The position of the body during rotation, i.e., whether it is upright or horizontal, is an important factor because of the effects upon the circulation, but, chiefly perhaps, because of the necessity of acts of equilibration mediated by the otoliths when the body is erect. Thus rotations about the long axis of the body with the subject sitting upright were very effective in causing sickness and distress and circulatory disturbances, whilst with the subject lying horizontally, rotations in the sagittal and coronal planes caused lighter and more transient phenomena. It was notable, however, that rotation in the sagittal plane backward, i.e., occiput first, caused much more disturbance than rotation in the same plane forward, i.e., face first.

For convenience, all rotations are to be considered as about the long axis of the body, with the subject sitting upright in an arm-chair unless otherwise specified.

In general, the effect of mild rotation upon the circulation is a rise in blood-pressure dependent on contraction of the blood-vessels. (See protocols 1-17 at the end of this chapter.) Frequently the rise is preceded by a preliminary fall in blood-pressure. If mild rotations be continued at frequent intervals, the rise in blood-pressure may occasionally be absent, owing to fatigue, but on resting, and at times even without resting, it will manifest itself soon again. After repeated mild rotations, a close study of the circulation reveals the fact, not fairly represented in the protocols, that the blood-vessels are constantly contracting and dilating. In severe rotations, the radial artery becomes at times so small and empty as to be impalpable. Under such circumstances, of course, the blood-pressure by clinical methods would be nil. During such periods of radial pulselessness, the ear, or stethoscope, placed over the heart found the latter beating, with the sounds feeble, the second sound being relatively accentuated. (See protocol 13). These pulseless periods occurred mainly in very rapid rotations during the actual rotation, and frequently just after reversal or sudden cessation. They lasted for a moment or two when the pulse gradually returned. At the time that the pulseless periods were observed, the swing used consisted of a boatswain's chair. There was, consequently, no support for the back, so that the subject's head tilted backward during the rotation. In later trials when the swing used (an arm-chair) had a support for the subject's back, no pulseless periods were observed. An approach to this phenomenon was frequently observed, both during and after rotations, when the pulse, at one moment large and with low blood-pressure, would almost immediately disappear as the arteries contracted to an extreme degree. (See protocol 13.)

After long, repeated, mild rotations, or after severe rotations with reversals or retardations, the general tendency is toward a decline in blood-pressure, with general prostration and with the pulse-rate in general stationary, or somewhat slowed. Vomiting restores the circulation so regularly in rotation sickness that it may be regarded as one of nature's defences against failing medullary circulation, rather than as a mere act intended to empty the stomach, though in doing the latter it unquestionably benefits the general condition materially. At times the blood-pressure and pulse-rate made sharp upward excursions before the vomiting actually occurred, and before the subject was aware that the abdominal muscles had become fixed in the act. This fact, not generally admitted by physiologists and clinicians, has been frequently observed by the author in other conditions. The increased blood-pressure seems to be caused by the same agency that sets the mechanism of nausea and vomiting going, and is not merely the result of muscular contraction. In general, the respiratory rate was increased during, and slowed after, rotation. Between rotations, the rate was slowed or about normal, never much above it. Severe and even mild rotations caused complete apnœa for short periods, followed by deep inspirations. (See protocols 8 and 10.) As a rule, the respiratory rate increases as the blood-pressure falls, and vice versa. When vomiting and deep breathing did not suffice to maintain or restore the blood-pressure, the pulse-rate occasionally became markedly increased, but increase in pulse-rate apart from vomiting was the exception, not the rule; in fact, the most striking general effect of repeated rotations upon the pulse is a slowing of the rate.

It was noted that covering the eyes and head had no appreciable effect in preventing the nausea and circulatory disturbances that attend rotations. (See protocol 2.)

Rotations in the sagittal and coronal planes, with the subject lying horizontally, lowered the blood-pressure. (See protocols 17a and 17b.) The pulse-rate was also lowered. After the rotations, the blood-pressure rose somewhat, but not to the extent observed following rotations about the long axis with the subject sitting erect.

The conclusions are:

1. That rotations in the upright position about the long axis of the body, affect the circulation by irritation of the medullary vaso-constrictor centres. There are reasons for believing that the semicircular canals are not in direct relation with the medullary vaso-constrictor centres, and that the rise in blood-pressure that accompanies rotations, etc., is the result of the cerebral and cerebellar disturbances responsible for the vertigo, displacements of equilibrium, etc. On the other hand, the otolithic apparatus seems to be in direct relation with the medullary vaso-constrictor centres. These structures appear to be the chief factors in the mechanism which regulates the blood-pressure when the subject assumes the erect posture. However, inasmuch as rotations probably affect the otolithic apparatus of the vestibule, as well as the ampullary receptors of the semicircular canals, it is probable that the otoliths are an important factor in the mechanism involved in the circulatory changes observed.

2. That by vaso-constriction, the blood-pressure is raised without a corresponding increase in pulse-rate, but when the rotations are frequently repeated over long periods, or when they are severe, with violent reversals or retardations, fatigue rapidly sets in, with failure of the circulation which, when profound, is met chiefly by enhanced respiratory movements and by vomiting. The latter tends to restore the circulation in the vital medullary centres, and removes the stomach contents.

3. That the vagus (cardiac inhibitory) centre is stimulated, as evidenced by the slow pulse, and in certain extreme conditions is so irritated as to practically bring the heart to a standstill for a time.

4. That the respiratory centre is inhibited, making the rate lower, and causing, at times, complete apnœa. The subsequently increased depth of the respiration is to be attributed in part to a prolonged period of latency and accumulation of CO_2 in the blood.

5. That the respiratory as well as the pulse-rate, accompanies the blood-pressure in the sharp rise of the latter that occurs with vomiting.

6. That in rotation in the sagittal and coronal planes, with the subject lying horizontally, the vaso-constrictor

action with its resulting rise in blood-pressure, is wanting, not because the rotation does not stimulate the medullary vaso-constrictor centres, but because the stimulation of those centres is insufficient to cause appreciable effect on the blood-pressure on account of the diminished tonicity of the vessels and lowered pulse-rate that obtain in recumbency, these being due, in part, to the inactive state of the otolithic apparatus.

7. That in rotations in recumbency, the vagus centre is stimulated, as manifested by lowered pulse-rate, which is masked, to a certain extent, because of the lowered pulse-rate induced by recumbency.

8. That a condition simulating surgical shock may be present with extremely contracted arteries. This seems to favor Porter's view of the mechanism of shock, viz., arterial constriction, or Henderson's view, viz., venous relaxation, as opposed to Crile's view, which attributes shock to arterial relaxation.

9. That the emptying of the stomach is but one of the bye-results of vomiting, where the latter occurs as the result of anæmia and asphyxiation of the medullary centres in profound circulatory depression from whatever cause. Thus, vomiting is to be regarded as a response of the organism analogous to that which occurs in an animal upon which artificial increase of intra-cranial pressure is practised. In the latter instance, asphyxia occurs first, then follows arterial constriction, with contraction of the muscles all over the body, e.g., increased peristalsis, arterial constriction, etc., phenomena which are fairly represented in the act of vomiting as seen in rotations. Vomiting, therefore, or the condition that immediately precedes it and is responsible for the actual expulsion of food from the stomach, is to be regarded as a defence of the organism against threatened dissolution from lowered blood-pressure, and consequent asphyxia of the medullary centres.

The Influence of Drugs upon the Circulatory Changes that Occur with Rotations

Strychnin and atropin in combination had the effect of maintaining the blood-pressure without appreciable change in severe swings with violent reversals. Protocol 3 shows how this was effected in part, i.e., by increased pulse-rate. The sensitiveness of the cardiac mechanism to altered conditions of intra-cardiac pressure is also well known. Slight cardiac arhythmia was also noted at times. The action of strychnin alone is shown in protocol 3. The blood-pressure was not so well maintained, nor were the effects upon the subject's general condition so favourable as when the combination of atropin and strychnin was used. Vagus stimulation effects are seen just after the rotation. It is fair to add that the subject's body was in a state of fatigue from the. effects of a severe handball contest, engaged in on the previous day, without proper preparation. Protocol 8 shows the effect of atropin, which tends slightly to prevent the marked fluctuations of the respiratory rate incidental to rotations. Protocol 9 shows the effect of atropin in maintaining a good average standard of blood-pressure. The subject (B), on whom the experiments in this instance were performed, was extremely sensitive to rotation. The severe swings given after the atropin could not have been tolerated without the drug. It should be noted, however, that the stomach was empty, which is at times an important factor in rotation sickness. The pulse-rate was not quickened in the rotations after atropin. This shows that the action of the drug on the cardiac vagus terminals was insufficient to overcome the effects of central vagus stimulation. The blood-pressure was well maintained by arterial constriction under the influence of atropin. Protocol 10 shows, on a different subject (S), practically what was shown in protocol 9. It also shows the tendency of the respiratory rate to rise as the blood-pressure falls, and to fall as the latter rises. Protocol 11 shows the effect of nitroglycerin, both before and after atropin. Before atropin had been given, the typical nitroglycerin effect was not a conspicuous feature, being overshadowed by the vaso-constrictor effect from the rotations, and this, notwithstanding that the subject felt the usual nitroglycerin effects in the head. Nitroglycerin, administered after atropin, shows a marked rise in pulse-rate, illustrating one of nature's methods in combating lowered arterial tension. The large fluctuations of pulse-rate and blood-pressure occurring between the administration of the atropin and the second dose of nitroglycerin are to be attributed to exhaustion of the neuro-vascular mechanisms, which was, in turn, the immediate reactionary effect of the struggle to maintain the blood-pressure, in spite of the relaxed musculature of the arterioles caused by the first dose of nitroglycerin. Protocol 12 shows the effects of strychnin and morphin. The pulse-rate was not increased, the blood-pressure being well maintained by vaso-constrictor action, and yet the rotations made the subject feel very dizzy, sick and nauseated. With these there were motor and mental depression and a general feeling of wretchedness. Vomiting did not occur, because the rotations were suspended in order to forestall it, and to relieve the subject's wretchedness, which persisted for over four hours, partly, perhaps, because of the well-known excretion of morphin into the stomach, protracting the period of nausea pending its reabsorption or elimination. Protocol 13 shows the effect of morphin when used alone. The drug was administered after many rather severe rotations, and at a time when there was a steady decline in blood-pressure, with a tendency of the pulse-rate to rise. Rotations after the morphin showed a fairly well-maintained blood-pressure without increase of the pulse-rate. Forty-two minutes after the administration of the morphin the subject was given gr $^1/_{80}$ of atropin crystals. Fourteen minutes after the atropin had been given, rotations so sickened the subject that he vomited. The blood-pressure and pulse-rate failed to show the usual atropin effect. With the vomiting, there was the usual sharp rise in blood-pressure and pulse-rate. A repetition of the rotation caused depression of the blood-pressure and pulse-rate, and the subject felt nauseated, dull and heavy. During a rotation, given 26 minutes after the administration of atropin, the pulse-rate at the wrist disappeared and the second sound of the heart was feeble or missing. The pulse disappeared frequently in the rotations given on this occasion, both before and after the administration of the drugs.

The effect of bromid of potassium is shown in protocol 14a. The rotations sickened the subject and caused vomiting. Between rotations, the blood-pressure was fairly sustained. It can be seen, however, that the pulse showed rather a tendency to slowing. The figures do not fairly represent the behaviour of the circulation, as extensive fluctuations occurred, which were so evanescent that there was not time to catch and record them. After vomiting, the efficiency of the circulation was enhanced. With bromid, although the subject was sickened quickly, there was undoubtedly less psychic wretchedness than was noted in rotations without the use of drugs, whilst between rotations there was absence of the usual dread of the next rotation.

The effect of digitalon is shown in protocol 14b. The subject felt his susceptibility increased, if anything, for the sickening effects of rotations. The blood-pressure showed a downward tendency, as did the pulse-rate after an initial rise. The first rotation was performed two hours and eighteen minutes after the administration of the drug, which was not sufficient time for a digitalis preparation, given hypodermically, to show its characteristic effects on the circulation. The digitalon caused some local irritation and edema, and a distinct increase of stomach irritability. The effect of hyoscyamin is shown in protocol 15. The drug evidently diminished the sickness and wretchedness that usually accompany rotations. There were wide fluctuations in the blood-pressure and in the pulse-rate. On the whole, with hyoscyamin the blood-pressure was not so well maintained as with atropin, and it is evident that the drug was not so potent as atropin in eliminating the effects of vagus stimulation, since the pulse showed no tendency to increase in rate, and was slowed, if anything, after the rotations. The drug was also found inferior to atropin in warding off stomach sickness, although it helped to a considerable extent in this direction, for the rotations were rather severe, with many reversals. The psychic and motor exaltation noted with atropin were missing. During some of the rotations an almost impalpable radial pulse was found, accompanying an accentuated second sound. This was found repeatedly. Of course, taking blood-pressures under the circumstances was out of the question. Protocol 16 shows the effect of bromid in a subject very susceptible to the effects of rotation. Whilst the latter tended to sicken, there was an absence of the usual wretchedness and psychic depression. There was no fear of being made sick, and the thought of rotations to come did not worry. It was noted that in rotations after bromid the tendency to nausea, so readily excited by the odour of tobacco-smoke, was somewhat lessened. The blood-pressure and pulse-rate showed effects similar to those shown in protocol 14. The effect of hyoscyamin is again shown in protocol 16.

The train of symptoms set up in rotation sickness persists till physical and mental depression ensues. The stomach, once disturbed, ever remains a source of secondary irritation to the medullary, cerebellar and cerebral centres. Hyoscyamin counteracts or eliminates the effects of this secondary irritation by depressing the sensory nerve terminals in the stomach. The drug, however, also depresses the psychic areas in the cerebrum. The bromides act merely by depressing the cerebral psychic areas, thereby eliminating the element of apprehension and psychic distress that is such a constant symptom in rotation sickness. Atropin has the advantage over bromides and hyoscyamin, in that it depresses the sensory nerve endings in the stomach and all over the body, and at the same time stimulates the psychic and motor areas of the cerebrum, as well as the respiratory and vaso-constrictor centres in the medulla. The protocols clearly show that atropin has a more potent effect than hyoscyamin in tending to paralyze the vagus nerve endings in the heart. However, a relatively larger dose of atropin was used.

The effects of atropin and strychnin in combination are shown in protocol 17, where the rotations were performed with the subject lying horizontally. The effects are similar to those noted in the rotations performed with the subject sitting upright. Note the reflex slowing of the pulse induced by recumbency, and the slowing induced by vagus-centre irritation from the rotation, and counteracted by atropin later on. (See protocol 3.)

These observations show:

1. That the combination of strychnin and atropin is more potent than either of these drugs used alone, and is

superior to any other drug or combination of drugs so far tried in combating the effects of rotation upon the stomach and cerebrum, and upon the mechanisms of the circulation.

2. That atropin, hyoscyamin and bromides are efficient in averting or ameliorating rotation sickness in the order named, atropin, however, being far more potent than hyoscyamin, as the latter is more potent than bromides, although the circulation seemed to be better maintained with bromides than with hyoscyamin.

3. That morphin and nitroglycerin are a hindrance, rather than a help, in rotation sickness, and are contraindicated in conditions akin to rotation sickness, although with morphin the circulation was well maintained.

PROTOCOLS.

1.—On "S," a Subject with Fair Toleration. January 1, 1909.

Pulse-rate.	Blood-press.	Remarks.
72	120	Normal before rotations.
76	100	Just after rotation (long axis).
76	120	1 min. after " "
64	120	2 " " "
64	95	Just after rotation. Artery small.
64	120	1 min. after "
56	120	2 " "
60	115	3 " "
60	115	Rotation.
96	140	Vomited as left swing.
84	145	1 min. after rotation.
76	115	2 " "
72	105	3 " "
72	110	4 " "
72	105	5 " "
68	105	Rotation.
104	150	Vomited.
96	145	1 min. after rotation.
80	120	2 " "
76	115	3 " "
80	105	4 " "
80	100	5 " "
72	105	Feels chilly.
76	90	Went to stove.
72	110	
72	100	

Pulse-rate.	Blood-press.	Remarks.
72	105	
72	125	Danced to "warm up."
72	105	
72	115	
84	85	Just after rotation. Eyes open.
64	105	1 min. after rotation.
60	100	
60	95	2 " "
74	95	
76	95	3 " "
72	100	
76	95	4 " "
72	100	
72	105	5 " "
84	105	Rotation. Eyes closed.
100	115	
80	150	Vomited.
72	125	1 min. after rotation.
80	115	
80	105	2 " "
80	105	3 " "

2.—On "B," a Very Susceptible Subject. January 1, 1909

84	120	Normal.
84	115	
76	130	Standing in rowboat as latter was rowed in a circle.
88	135	Balancing efforts in cold air.
	130	No sickening effect.
76	120	Normal.
88	135	After mild to and fro swing in different directions.
88	140	
88	140	
76	130	Just after rotation long axis.
76	140	Nauseated.
72	125	After mild rotation.
68	140	1 min. after rotation.
72	140	2 " "
72	130	3 " "
72	130	Resting.
68	130	"
72	135	After mild rotation, eyes closed and covered.
72	125	Nauseated. Odours sicken.
72	140	
80	125	After brisk rotation.
68	120	Lump-sensation in stomach.
	140	Odour of tobacco-smoke offensive.

3.—On "S," January 2, 1909

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	76	120	Normal.
	76	115	"
	68	110	Mild rotation (long axis).
	72	115	1 min. after.
	68	115	2 "
	80	110	Mild rotation.
	88	110	1 min. after.
	84	110	2 "
	80	90	After brisk rotation.
	76	95	1 min. after.
	76	110	2 "
	72	95	After brisk rotation.
	56	90	Just after rotation, lying supine.
	56	95	
	64	110	1 min. after rot., lying down.
	72	115	Standing after lying. Feels chilly.
	68	100	1 min. after standing up.
	68	100	
	68	100	2 min. after standing up.
	60	100	
10:00			Strychnin sulph., gr 1/20, hypo.
	80	105	Resting.
	80	100	"
	72	100	"
10:10	68	90	Brisk rotation.
	64	95	1 min. after.
	64	95	
	64	95	2 min. after.
	72	115	3 "
10:20	68	100	Brisk rotation.
	60	100	1 min. after.
	64	110	2 "
	64	110	3 "
10:37			Atropin sulph., gr 1/100, hypo.
	60	95	Resting.
	64	105	"
10:43	76	105	"
10:45	72	105	Brisk rotation, dizzy during rot. no nausea.
	60	105	1 min. after.
	60	105	2 "
	68	105	3 "
	72	105	4 "
	80	105	6 "
	88	105	7 "
	84	105	8 "
11:00	92	105	Brisk rotation, dizzy; nauseated, headache.
	88	105	1 min. after.
	84	105	2 "
	80	105	3 "

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	88	105	4 min. after.
	88	105	5 "

4a.—On "S," January 3, 1909

	72	110	Normal, standing.
	68	105	" "
	56	95	Lying.
	52	95	"
	56	100	Standing.
	72	110	"
	72	110	After mild rotation (long axis).
	68	115	" " "
	68	105	" " "
	60	105	" " "
	64	100	" " "
	64	95	" " "
	72	95	" " "
	64	105	" " "
	60	95	" " "
	64	95	" " "
	64	95	" " "
9:55			Atropin sulph., gr 1/100, hypo.
	64	95	
9:57	64	100	
	56	100	
10:00	60	100	
10:02	64	105	
10:05	64	100	
10:09	64	100	
10:10	80	100	Brisk rotation; slight momentary nausea.
	80	100	Nervous; dizzy; weak at knees.
	76	105	
	72	105	
	84	105	
	88	100	
	88	95	
	84	95	Lay down.
	56	95	Lying down.
	64	100	"
	68	105	"
	64	105	"
	92	95	Standing after lying.
	92	95	" "
		95	" "
	96	95	After a moderate rotation.
	80	95	
	88	100	" " "
	84	100	" " "
	80	95	" " "

4b.—On "B," January 3, 1909

Pulse-rate.	Blood-press.	Remarks.
76	115	Normal, standing.
76	115	"
76	110	Lying.
72	115	"
80	110	Standing.
80	110	"
92	115	After a moderate rotation.
80	110	" " "
80	110	" " "
76	115	" " "
80	115	" " "

5.—On "S," January 10, 1909

Pulse-rate.	Blood-press.	Remarks.
72	110	Normal, standing.
68	110	"
76	115	"
76	115	"
76	115	Lying down.
60	95	"
64	105	"
60	95	"
60	105	"
60	105	"
64	105	"
60	100	Standing.
68	110	"
68	100	"
72	105	"
76	110	"
72	105	"
72	110	"
68	120	After rotation (long axis).
68	120	"
68	115	2 min. after.
68	115	3 "
68	110	After a moderate rotation.
64	105	" " "
68	105	" " "
64	110	After a brisk rotation.
60	95	1 min. after.
60	95	2 "
64	100	3 "
64	95	4 "
68	95	
68	120	Hanging by legs, trapeze fashion.
60	125	" " "
64	130	" " "

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Pulse-rate.	Blood-press.	Remarks.
68	95	Standing.
68	105	"
68	105	"
64	105	"

6.—On "S," January 10, 1909

72	95	Sitting.
72	95	"
72	100	"
84	110	Standing.
88	115	"
84	115	"
64	100	Lying supine.
64	105	"
64	105	"
64	130	Head lowered; body making angle of 30° with horizon. Pupils contracted; face congested; sense of pressure over forehead.
64	110	Head still low.
64	110	"
64	105	"
64	110	"
68	110	"
92	105	Standing up after lying with head low; pupils dilated, face pale, radial artery contracted. On standing up blood-pressure went up to 125 or above it, but immediately fell to 105.
84	105	Standing after lying with head low.
84	110	" " "
88	115	" " "
84	115	Head low. Body making 30° with horizon. Pupils contracted, face congested.
56	90	Lying head low.
60	95	"
60	95	"
60	95	"
80	125	On sudden throwing of subject into sitting posture.
84	110	Sitting after lying with head low. Pupils moderately dilated, radial artery small.
84	105	Sitting after lying with head low.
84	105	Radial artery larger.
84	120	Laughed; artery smaller.
80	115	Sitting.
84	110	"
84	105	"
84	105	"
84	125	Standing, after sitting.
80	115	" "

Pulse-rate.	Blood-press.	Remarks.
84	105	Standing, after sitting.
80	95	" "
76	110	" "
80	110	Standing.
80	110	"
60	90	Lying. Pupils small; face congested; radial artery dilated.
64	105	Lying.
60	105	"
60	100	"
60	100	"
64	90	Lying head low. Pupils small, etc., as before.
60	100	" " "
56	100	" " "
60	95	" " "
60	100	" " "
60	95	" " "
80	105	Standing.
84	105	"
84	95	"
80	110	"
80	105	"

7a.—On "B," January 10, 1909

72	120	Normal, standing.
76	115	" "
76	110	" "
76	110	" "
76	130	After mild rotation (long axis).
72	125	1 min. after.
72	120	2 "
80	125	After a brisk rotation.
76	120	1 min. after.
76	120	2 "
84	115	After brisk swing. Dizzy, nauseated, apnœa, followed by deep inspirations.
72	110	1 min. after rotation.
68	105	2 " "
68	100	3 " "
68	110	4 " "
68	105	5 " "
68	100	After mild rotation. Dizzy, nauseated. On verge of vomiting.
68	105	1 min. after rotation.
64	105	2 " "
60	115	After mild rotation. Pulseless at wrist after rotation. On verge of vomiting.
68	110	1 min. after rotation.
64	115	After mild rotation.

Pulse-rate.	Blood-press.	Remarks.
68	105	1 min. after.
68	100	After mild rotation. After a few turns, was on verge of vomiting.
68	110	1 min. after rotation.
68	105	2 “ “
68	100	After mild rotation.

Pulseless at wrist for a moment or so after rotation. After this rotation the subject did not recover as after the previous rotations, but remained sick and wretched and became yellowish green in the face. The odour of tobacco-smoke was offensive. Vomiting did not occur, as the rotations were discontinued to avert it. It took an hour or more before the subject's stomach ceased to distress him.

7b.—On “B,” January 17, 1909

84	125	Normal, standing.
84	125	“ “
76	125	“ “
72	120	Lying.
72	110	“
88	115	Standing.
88	120	“
88	120	“

8.—On “B,” January 24, 1909

Respirations.	Remarks.
16	Normal, standing.
16	“
18	“
16	Normal, lying.
16	“
18	Standing.
14	“
16	“
14	“
14	“
16	“
14	Just after a mild rotation.
16	1 min. after.
18	2 “
14	3 “
18	After mild rotation.
16	1 min. after.
16	2 “
18	After mild rotation.
16	1 min. after.

Time, a.m.	Respirations.	Remarks.
	14	2 min. after.
	16	3 "
	14	After rotation.
	18	1 min. after.
	16	2 "
	20	After brisk rotation.
	16	1 min. after.
	16	After rotation.
	12	1 min. after.
	8	2 "
	16	3 "
	12	After rotation. Left swing vomiting.
	16	1 min. after.
	20	2 "
	18	3 "
	18	4 "
	16	5 "
	12	6 "
10:02	16	Atropin sulph., gr 1/100, hypo.
10:15	18	
10:33	16	
10:34	14	
10:35	14	After brisk rotation.
	14	1 min. after.
	12	2 "
	14	3 "
	16	4 "
	14	5 "
	14	6 "
	16	7 "
10:45	18	After long, brisk rotation.
	14	1 min. after.
	18	2 "
	16	3 "
	16	4 "
	14	5 "
	16	6 "
	12	7 "
	16	After climbing rope, hand over hand.
	16	1 min. after.
	14	2 "
	14	3 "

Note.—Apnœa preceded the vomiting. The rotations given after the administration of atropin drove the respirations down to 12 or 14, whilst at no time after the drug were the respirations higher than 18, the average being about 16. Even brisk and prolonged rotations caused no greater fluctuation in blood-pressure than 5 mm, whilst the pulse-rate never rose above 70, the average being about 64, with a minimum of 56. After atropin, the rotations failed to sicken the subject.

9.—On "B," January 24, 1909

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	76	120	Standing.
	84	115	"
	76	115	"
	72	130	Lying.
	72	130	"
	68	115	"
	68	115	Standing.
	68	115	"
	72	110	"
	72	150	Hanging by groins on trapeze.
	92	125	Standing.
	72	125	"
	72	115	"
	72	110	After mild rotation.
	68	95	1 min. after.
	72	120	After mild rotation.
	72	110	1 min. after.
	76	115	After mild rotation.
	76	115	1 min. after.
	76	125	After brisk rotation. Lump-sensation in stomach.
	72	115	1 min. after.
	68	110	After mild rotation; sick feeling; muscular weakness.
	76	110	1 min. after.
	80	135	After mild rotation. Left swing vomiting.
	76	130	1 min. after.
	72	120	2 "
	72	120	3 "
	72	115	4 "
10:02	72	115	Atropin sulph., gr 1/00, hypo.
	68	115	Resting.
	68	115	"
	68	115	"
	68	110	"
10:12	60	115	"
	64	115	"
	68	115	"
	68	115	"
	64	120	"
	64	120	"
10:45	60	110	After long brisk swing. Practically no dizziness or sickness. Felt strong.
	56	120	1 min. after.
	56	110	2 "
	64	110	3 "
	68	105	4 "
10:45	60	105	After brisk rot. No sickness or discomfort.
	60	115	1 min. after.

Pulse-rate.	Blood-press.	Remarks.
60	115	2 min. after.
60	110	3 "
72	125	After climbing rope, hand over hand.
68	125	1 min. after.
64	125	2 "
64	120	3 "

The rotations given the subject were exceedingly mild and of short duration. With the third rotation, symptoms of rotation sickness set in, but passed off almost immediately. The familiar lump-sensation referred to the stomach appeared with the fourth rotation. After this rotation the respirations were slowed to 8, but immediately returned to 16. With the sixth rotation, the subject began to feel "sick all over." The stomach sickness subsided somewhat immediately, but weakness and tremulousness in the limbs persisted. In the seventh rotation the subject left the swing vomiting. The vomiting at once relieved depression and distress; the head became clear and, instead of being dull and morose, the subject became cheerful and talkative. The vomitus contained a large quantity of thick mucus and some food. The vomiting was of the projectile variety, and was followed by a sharp, crampy pain in the epigastrium. For some time before the onset of vomiting, there was a profuse flow of saliva.

After atropin rotations had little effect in causing even momentary distress. It should be noted, however, that the stomach had already been emptied by the previous vomiting. The slow pulse after atropin was remarkable. Even rope-climbing, hand over hand, did not appreciably accelerate the pulse. Under ordinary conditions, such exertion would send the subject's pulse-rate up to 90 or 100, or even higher.

10.—On "S," January 31, 1909

Pulse-rate.	Blood-press.	Remarks.
76	115	Normal, standing.
76	115	" "
80	110	" "
88	110	" "
84	115	" "
84	110	" "
80	110	" "
80	110	After mild rotation.
72	120	1 min. after.
76	120	2 "
72	130	After rotation.
72	120	1 min. after.
84	115	2 "
76	110	3 "

Time, a.m.	Pulse-rate.	Blood-press.	Respirations.	Remarks.
	80	110	..	4 min. after.
	76			Pulseless momentarily after rotation.
	72	115	..	After hard rot. Dizzy. Artery small.
	76	105	..	1 min. after.
	72	110	16	2 "
	72	120	16	3 "
	72	110	18	4 "
	72	100	20	After rot. Dizzy; lump-sensation; sick.
	68	110	18	1 min. after.
	72	115	16	2 "
		90	18	After rotation.
		120	16	1 min. after.
	72	110	16	2 "
	72	120	18	3 "
	72	110	16	4 "
		100	18	After rotation.
	68	120	16	1 min. after.
	68	115	16	2 "
	72	105	14	3 "
		100	16	4 "
	68	100	14	5 "
	76	100	18	Standing.
	68	95	..	"
10:21				Atropin crystals, gr 1/60, hypo.
10:50	88	95	..	After standing near stove.
	72	95	..	Feels well.
	72	95	..	"
	72	95	..	"
10:53	68	120	..	After hard rotation.
	64	120	..	Momentary dizziness during rotation.
	64	115	..	No sickness or distress.
	68	115	..	

Note.—In general, the respiratory rate rises as the blood-pressure falls, and *vice versa.*

11.—On "S," February 7, 1909

Pulse-rate.	Blood-press.	Remarks.
72	110	Normal, standing.
72	110	" "
68	115	" "
72	115	" "
72	115	" "
68	115	After hard rotation. Dizzy. Lump in stomach. Saliva free.
68	120	1 min. after.
68	110	2 "
72	115	3 "

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	68	115	4 min. after.
	72	95	After hard rotation. Feels like vomiting. "Gone feeling."
	68	105	1 min. after.
	72	95	2 "
	68	110	3 "
	72	105	After a rest. All symptoms gone but "lump" in stomach.
	68	105	

After rotation nervous. Feels like vomiting. Odour of alcohol disagreeable. Pulse varies from moment to moment. Yawning. Saliva profuse. Head clear.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
		100	
		105	1 min. after rotation.
	68	110	2 " "
	76	105	3 " "
		90	4 " "
		105	5 " "
	72	115	6 " "
		90	7 " "
		105	8 " "
	68	105	9 " "
9:29			Nitroglycerin, gr 1/100, hypo.
	64	95	After nitroglycerin.
	68	100	" "
	68	110	" "
	68	100	Throbbing sensation over forehead.
	68	105	Sick feeling in stomach.
	76	110	No nausea. Feels chilly.
	76	100	
9:37	72	100	After rotation pulse did not disappear.
9:38	76	115	Feels like vomiting. Feels exhausted
9:39	76	105	Headache, nausea.
9:39½	76	115	
9:40	76	110	Feels well, but has frontal headache.
9:41	76	105	After rot. Sick, nervous, chilly, shivering.
9:42	72	110	Artery large. Pulse easily felt, but of Corrigan type.
9:43	72	105	
9:44	72	105	Head clear.
10:03			Atropin crystals, gr 1/60, hypo.
10:12		80	Wretched and shivering. Hard to find radial artery. Heart's first sound long and booming.
10:13	80	90	
10:17	96	105	After exercise (dancing) pupils contracted.
10:18	76	105	Headache (vertex). Lump in stomach,
10:19	84	95	Head has not cleared yet.
10:20	88	95	

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
10:30	80	95	Pain in forehead and in left of epigastrium. This latter was present before the experiments were begun.
10:31	88	95	
10:32	88	105	After rotation. Pulseless at first. No nausea. Feels well.
10:33	88	85	Pulse comes and goes. Artery very small.
10:34	88	80	
10:42			After rotation. No bad effects. No pressures taken.
10:47	92	95	Feels very well. Talkative. Resting.
	88	110	
	80	115	
	88	115	
10:52	84	105	After rotation. Pulseless at first.
	84	100	Momentary dizziness. No nausea or tendency to vomit.
	72	105	
10:57			Pulse varies markedly in force, and at times seems to disappear.
10:58			Nitroglycerin, gr 1/100, hypo.
10:58½	84	85	Feels well.
10:59	80	90	Headache returning.
11:00	96	90	
11:01	92	95	
11:02	88	95	
11:05	84	95	After rotation. No sickness or distress from rotation.
11:06	92	105	
11:07	92	105	Pulse did not disappear.
11:08	96	105	

Note.—Nitroglycerin prevented marked fluctuation of the blood-pressure after rotations, possibly by its effect in maintaining equalized intra-cardiac pressure. Atropin, by counteracting the dilatation of the arterioles, caused the fluctuations in blood-pressure to reappear. After the second dose of nitroglycerin the fluctuations in pressure were again partially suppressed.

12.—On "B," February 12, 1909

80	120	Normal, standing.
76	120	" "
76	120	" "
76	110	" "
		After rotation dizzy, fulness in head, chilly, momentary pulselessness.
76	105	1 min. after.
76	115	2 "

Three rotations were given to note the variations of the pulse and the accompanying symptoms. During the first, which was a mild rotation, the pulse disappeared and returned many times. There was no nausea or distress in the head at first. After a time the lump-sensation was felt in the stomach, and with it fulness in the head and dizziness. Vertigo was aggravated by looking up over head, but not on looking at things straight in front. During the second rotation the pulse did not disappear, but the artery became extremely contracted, with apparently well-maintained blood-pressure. The subject was nauseated and on the verge of vomiting. These symptoms disappeared when the swing was stopped. In the third rotation it was found that the effects of rotation could be better resisted if the eyes were kept closed. Opening the eyes during a rotation precipated the subject's sickness. During the rotation the pulse did not disappear, but afterward the artery became very small and at times the pulse could not be felt. When the pulse returned, the artery was dilated and the pressure low. During the rotation there was a marked tendency to vomit. After the rotation recovery was slow and incomplete.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	80	100	After rotation. The pulse persisted through the rotation, but the pressure seemed to be low. Resting the head against the rope retarded the sickness. The subject was very wretched on the swing, had frequent eructations, and was on the verge of vomiting. After the rotation the subject felt weak and wretched and wanted to lie down, or even to put his feet somewhere to rest them.
9:45	..	..	Strychnin sulphate, gr 1/30, hypo. The subject felt no pain from the hypo. Recovery was slow after the last rotation. The subject felt weak and disinclined for work. He felt loss of control of the movements of the head when the latter was tilted from side to side. Flatus was passed.
9:52	..	..	Knee-jerks slightly overactive. Achilles jerk absent. The subject went and stood by the hot stove. This caused return of the lump-sensation and stomach distress.
10:11	64	100	Normal.
10:12	64	100	"
10:13	64	105	After rotation.
10:18	..	..	Morphin sulphate, gr 1/6, hypo.
10:30	64	130	Head clear. Feels well. Pupils contracted.
10:31	68	135	
10:32	64	130	
10:33	64	130	
10:34	64	130	

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
10:35	72	130	After rotation. Nausea. Lump-sensation. Muscular inefficiency.
10:36	72	125	1 min. after.
10:37	76	120	2 "
10:38	76	120	3 "
10:46	68	120	After mild rotation.
10:47	68	120	
10:48	68	120	Subject became green in the face and very sick. Markedly nervous and weak, especially in the legs. Brain tired and clouded. On the verge of vomiting.

The rotations were discontinued to avert vomiting. This probably had much to do with prolonging the subject's sickness, for it was fully four hours before the subject felt himself again.

It is evident that the immediate effects of rotation sickness are not dependent on circulatory impairment; for, in spite of well-maintained pulse-rate and blood-pressure after the administration of morphin, the susceptibility of the subject to rotation sickness was enhanced.

13.—On "S," February 14, 1909

76	115	Normal, standing.
72	115	"

Two rotations were given without taking observations on the pulse-rate and blood-pressure. In the first rotation there was slight dizziness, and the pulse almost disappeared, but gradually returned with full force. In the second rotation the pulse did not disappear, but the artery was much constricted. The heart-sounds were normal, the second aortic having a well-marked click. There was some dizziness, with increased salivary flow. Recovery was prompt.

68	110	Normal, standing.
68	115	"

Two rotations were given with the eyes closed and covered, and with the auditory canals plugged tightly. The first was a hard, brisk rotation. The pulse did not seem to change during the rotation. There was no dizziness nor distress. Only a soft, soothing motion was experienced, though the saliva was profuse. During the second rotation the pulse almost disappeared, but soon returned with full force and constricted arteries. The rate at this time was 68. The salivary flow was profuse, and there was dizziness and commencing lump-sensation in the stomach. When the swing was stopped the subject felt that he was still turning. Tactual impressions were the only means he had of knowing whether he was moving or not. The radial artery was dilated, and its wall quickly receded after the upstroke of the pulse, suggesting the Corrigan pulse. The subject felt himself getting sicker and sicker.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	64	115	2 min. after rotation.
	72	110	3 "

Two rotations were given with the eyes closed and the ears plugged. In the first rotation, which was a mild one, the subject experienced dizziness and lump-sensation in the stomach. He yawned much, and the salivary flow was profuse. In the second rotation the pulse did not disappear, but it became very small and scarcely perceptible. The second aortic sound was lengthened and loud. The first sound seemed feeble. The upstroke of the pulse was feeble. Three minutes later the rate was 64, and the blood-pressure 115, and then the second aortic sound was clear and distinct as compared with one minute previously, when the pulse was feeble and the second aortic sound almost inaudible. There were quick and wide-ranging variations in the blood-pressure. The subject felt chilly along the spine, and yawned frequently. The salivary flow was profuse. No lump-sensation was experienced, but there was a constant tendency to "gag."

	64	115	3 min. after rotation.
9:52	..	..	Morphin sulphate, gr 1/6, hypo.
9:53	72	110	Chilly and nervous. Some nausea.
9:55	72	100	Artery small. Lump sensation.
9:57	76	110	Eructations. Pain in epigastrium.
9:59	72	100	Numbness in hands. Feels tired.
10:01	68	95	First heart-sound feeble and short, with indistinct second sound. Feeling of tightness across forehead. Slightly nauseated.
10:04	68	100	After rotation.
10:05	68	105	

During the rotation the first heart-sound was scarcely audible. The second sound was fairly audible, but there was no snap at the closure of the valves. The pulse did not disappear, but it became very feeble. The subject became dizzy and nauseated, and was on the verge of vomiting. Recovery was much retarded as compared with other rotations.

10:07	68	105	
10:12	68	115	The radial artery was not so constricted as previously. The first heart-sound was feeble. There was no pain nor lump-sensation in the stomach. The subject felt well.
10:14			During a rotation there was slight dizziness, but no nausea or lump-sensation. The subject felt well, but "the brain was clouded." The pulse did not disappear. The artery was small, and the pressure well maintained.
10:15			During a rotation the subject was not made dizzy or sick, and it did not distress him to look at objects. The stomach felt well, but the saliva was increased.
10:18			A very brisk rotation was given. In this rotation the

subject's head extended backward so that his body approached the horizontal plane. During the rotation the subject felt no sickness or distress. The pulse disappeared at first, but soon returned. The heart-sounds were fairly normal, but the rate was considerably slowed. One minute after the rotation the subject began to get sick. The lump-sensation in the stomach returned and with it nausea. The subject became sicker and sicker, until his wretchedness became extreme.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
10:25	..	90	Nervous. "Heartburn."
10:26	..	95	
10:27	..	100	Brain clouded.
10:28	64	100	No lump-sensation.
10:29	64	105	After rotation. Artery extremely small. Dizziness. Nausea. Lump-sensation. On the verge of vomiting. Listless. Wants to lie down.
10:30	64	110	1 min. after rotation.
10:31	64	110	2 " "
10:34			Atropin crystals, gr 1/60, hypo.
10:35	64	95	Mist over eyes. Otherwise well. No nausea. No dizziness.
10:37	64	105	Feels better. Eyes clearing. Stomach feels well. No lump-sensation. Feels that he is recovering.
10:40	..	95	
10:41	64	90	Artery almost impalpable.
10:42	64	105	Artery small. Feels well, but has slight mist over eyes.
10:44	64	100	Does not feel well.
10:45	..	105	Feels nervous.
10:48			A rotation was given. The subject became dizzy and nauseated, and finally vomited. One minute before vomiting occurred the arteries became contracted and the blood-pressure rose to 120, but the subject said that there had already been some contractions of the abdominal muscles. After the vomiting the blood-pressure was 120, with extremely constricted arteries. The head felt light. The mouth was dry; the heart-beats were strong, but irregular in rhythm. The stomach felt well, but the subject felt nervous and had "heartburn."
10:50	80	115	After vomiting.
10:53	64	105	
10:54	68	105	
10:56	60	110	
10:57	68	110	
11:00			A brisk rotation was given. The pulse disappeared. The second aortic sound was inaudible at times, and

at other times scarcely audible. The subject experienced no dizziness, and only slight nausea. His voluntary muscular power seemed to be good. During the rotation, the eyes were the chief source of distress. He experienced some lump-sensation in the stomach at times, and at other times he was quite free from it. Recovery was somewhat retarded. The subject felt nervous.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
11:04	..	85	After rotation.
11:05	..	105	"
11:06	60	95	"
11:07	..	100	"
11:08	60	100	"
11:09	..	..	Feels dull and heavy. Has no nausea. Stomach feels well. Voluntary muscular power is good.

14a.—On "S," February 21, 1909

7:15	..	..	Bromid of potassium, gr lx., *per os.*
8:45	80	110	Normal, standing.
8:46	76	110	"
8:51			Rotation. Slight dizziness. Pulse did not disappear. Saliva increased. Eyes distressed subject.
	64	120	After rotation.
	72	120	"
8:55			Rotation. Very dizzy. Nauseated; salivary flow profuse. Pulse did not disappear, but arteries contracted and dilated. The blood-pressure varied. Pulse of the Corrigan type at times.
	68	120	After rotation.
	64	105	"
9:04			Rotation. Pulse did not disappear, but the radial artery became very small. Very dizzy. No lump-sensation. Eructations. Distress in eyes.
	72	120	After rotation.
	72	115	"
9:07			Rotation. Dizziness, heartburn, eructations. Pulse did not disappear. Rapid fluctuation in pressure, which was as low as 90 at times. The subject's distress quickly disappeared, although the fluctuations in the blood-pressure persisted.
	64	115	After rotation.
	68	110	"
9:12			Rotation. Very sick and dizzy. Eructations, flatus. Twitching in the left upper eyelid. Saliva profuse. Lump-sensation. Recovery quick. Pulse did not disappear, but was very small. Subject was chilly and felt as if going to vomit.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	68	115	After rotation.
	72	105	“
	68	115	“
9:17			Rotation. Nauseated and dizzy. On the verge of vomiting. Nervous. Pulse did not disappear.
	68	120	After rotation.
	68	120	“
	72	110	“
9:25			Rotation. Became nauseated and vomited. Subject felt his skin hot. Pulse did not disappear. Nervous. Head clear.
	80	125	After vomiting.
	80	115	“
	72	120	“
	72	120	“
9:33			Rotation. Slight dizziness. No sick feeling. Quick recovery. Feels a little weak and nervous, but this does not worry him. Stomach gives slight intimations of disturbance. Saliva about normal.
	68	120	After rotation.
	68	120	“
	68	110	“
	68	115	“
	68	115	“
9:40			Rotation. Nauseated, weak, and dizzy; chilly; nervous and weak. Feels as if there is gas in the stomach. Difficult to keep eyes open.
	68	120	After rotation.
	64	120	“
	64	120	“
	68	115	“

14b.—On “B,” February 21, 1909

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
7:40			Digitalin, minims xv., hypo.
7:45	72	125	
	80	120	
	76	120	
9:52	80	110	
	80	110	
	84	105	
9:58			Rotation. The subject was made very dizzy. When the swing was stopped he felt himself rotating in the opposite direction. The pulse did not disappear.
	88	110	After rotation.
	76	110	“
10:07			Rotation. Very dizzy. On the verge of vomiting. Weakness about the eyes and wretched feeling in head. The subject felt that he was more suscep-

tible than usual to the effects of rotation. The mere thought of the swing made him wretched. The pulse did not disappear. The time allowed between the administration of digitalin and the rotations was perhaps insufficient to secure full action of the drug.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	76	100	After rotation.

15.—On "S," February 22, 1909

9:15	76	115	Normal, standing.
	72	115	"
			Rotation. Radial artery dilated during rotation. Dizzy. Pulse did not disappear. On stopping the swing external objects seemed to move in the direction opposite to that of the rotation. Just after the rotation the radial artery became small.
	68	120	After rotation.
			Rotation. Dizziness, but no nausea or sickness. Pulse did not vary much.
	68	110	After rotation.
			Rotation. Dizzy, nauseated, lump-sensation, on the verge of vomiting. Saliva profuse. Recovery slow. Beginning to feel nervous. Pulse remained and was large and soft. After the rotation the radial artery contracted and dilated, and the blood-pressure varied from moment to moment, being at times as low as 90. The subject felt sick and wretched. He was chilly and nervous, yawning from time to time.
	68	125	After rotation.
	64	115	"
	64	115	"
	68	120	"
	..	110	"
	..	115	"
	..	110	"
	80	110	"
			Rotation. Very dizzy. On the verge of vomiting. Heartburn. Saliva profuse. Hiccough (after pressures were taken). Pulse became very small and hard. Eructations, lump sensation, grogginess.
	64	120	After rotation.
	78	105	"
	..	110	"
	..	105	"
	..	115	"
9:57	64	110	Normal, chilly, nervous.
	72	105	Pulse irregular in rhythm.
9:58			Hyoscyamin, gr 1/100, hypo.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
9:59	56	100	
10:45	..	..	Pulse irregular in rhythm, especially on deep inspiration. Feels chilly. Temperature of air 28° F.
10:46	52	100	Normal, standing.
	60	100	"
10:50			Rotation. Dizziness. Heavy feeling across forehead. Pulse slow and feeble. Pupils contracted. No nausea or sickness. External objects seemed to move about the subject's body in the horizontal plane.
10:54			Rotation. Very dizzy. Momentarily nauseated. No lump-sensation. No excess of saliva. Recovery prompt.
	52	100	After rotation.
	52	110	"
	56	110	"
11:00			Rotation prolonged and brisk, with reversals. Dizzy at reversals, but the feeling passed off immediately. Toward the end the subject felt as if he would fall out of the swing. Slight mist before eyes. Momentary nausea after each swing. After the rotations the subject had eructations and heartburn, and felt sick. In a moment, however, he felt well, but had intimations of an oncoming headache. He had a faint feeling in the stomach as if he had indigestion, but there was no accompanying feeling of wretchedness. He had not the usual feeling of exhaustion. He had no dread of the swing "because there was no feeling to it" after the hyoscyamin.
11:02	52	80	After rotation.
11:03	52	95	"
11:04	52	105	"
11:06	60	110	"
11:07	56	100	"
11:17			Rotation. As the swing stopped the subject was on the verge of vomiting. The usual symptoms were present, viz.: dizziness, heaviness in head, lump sensation in stomach, increased saliva. The pulse disappeared during the rotation, and the second aortic sound was almost inaudible. Recovery was prompt. The subject felt chilly, but was not nervous.
11:18	56	95	After rotation.
11:19	56	100	"
11:20	52	95	"
11:21	..	95	"
11:37			Rotation. Dizzy. Inclined to vomit. Lump-sensation. Recovery slow. Slight heartburn. Beginning to fear swing. During the rotation the pulse disappeared at the wrist, but the aortic second sound was more pronounced and snappy than before the rotation.

Time. a.m.	Pulse-rate.	Blood-press.	Remarks.
11:38	..	100	After rotation.
11:39	64	95	"
			The rotations given after hyoscyamin were long and brisk as compared with the rotations given before the hyocyamin, and yet the subject readily recovered from their effects. When the arteries were extremely contracted the blood-pressure apparatus was inefficient. An accentuated second aortic sound was repeatedly found to accompany an almost impalpable radial pulse.

16.—On "B," February 22, 1909

7:25	..	..	Potassium bromid gr lx., *per os.*
10:00	72	105	Normal. Watery, metallic taste in mouth.
	76	105	"
	72	105	"
10:04			Rotation. Dizziness and distress, especially referable to eyes. No sick feeling on reversals. On looking up and down, external objects seemed to move with a wavy up-and-down motion. Slight lump-sensation in stomach.
10:08			Rotation. Pulse showed little variation and did not disappear. During the rotation, looking overhead caused extreme dizziness. Lump-sensation. Recovery prompt, but subject is afraid of the swing.
10:10			Rotation. On the verge of vomiting, but no feeling of wretchedness and no dread of the swing. The subject was satisfied to go on with the rotations until vomiting should occur. Recovery prompt. Odour of tobacco-smoke not offensive. Lump-sensation referred to the stomach. The pulse did not disappear during the rotation, but the artery was contracted.
10:12	80	120	After rotation.
10:13	76	100	"
10:19			Rotation. Lump-sensation. On the verge of vomiting. Only a slight feeling of sickness. Not afraid of swing. No mental depression. Odour of tobacco-smoke nauseates. Eructations.
10:21	..	100	After rotation.
10:22	..	105	"
10:23	72	115	
10:24	..	110	
10:39			Hyoscyamin, gr 1/100, hypo.
11:39			Lump-sensation. Very dizzy, on the verge of vomiting. Recovery prompt, but lump-sensation persisted. Mouth not dry. Pulse 56 at 11:40, 11:42, and 11:43.

The rotations given after the bromid would, under ordinary circumstances, have sickened the subject. The absence of psychic depression and fear of the swing were noteworthy, and must be attributed to the effect of the bromid.

The rotation given after hyoscyamin was of the same duration and intensity as the rotations given the same subject on another occasion after atropin, when no disagreeable effects were suffered. It should be remembered, however, that on that occasion the subject had previously vomited. This fact tends to offset somewhat the apparent superiority of atropin in preventing rotation sickness.

17a.—On "S," May 21, 1909

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
9:56	64	105	Normal, sitting.
	68	105	"
	64	105	"
10:09	52	105	Lying on back in swing.
	56	105	" "
	52	105	" "
	52	105	" "
10:38	52	90	After double rotation in coronal plane.
10:54	52	90	" "
	48	90	1 min. after.
	48	95	2 "
	48	95	3 "
11:01	52	95	Resting.
	48	95	"
	48	100	"
11:04	44	95	After hard rotation in coronal plane.
11:05	44	95	" "
11:06	44	95	" "
11:07	44	59	" "
11:10	44	85	" "
11:11	48	90	" "
11:12	44	100	" "
11:13	44	105	" "
11:14	48	115	" "
11:18	48	105	" "
11:19	48	105	" "
11:20	48	105	" "
11:21	48	105	" "

17b—On "S," June 6, 1909

7:20	..	..	Atropin sulphate, gr 1/75, Strychnin nitrate, gr 1/40, both hypo.
8:40	120	110	After mild exercise.
	108	110	"
	108	110	"
	100	110	"

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
9:18	72	95	After rotation in coronal plane.
9:19	68	95	" "
9:22	64	105	" "
9:23	60	105	" "
9:27	60	95	" "
9:28	60	100	" "
9:30	60	105	Resting.
9:31	64	105	"
9:32	56	95	"
9:33	56	100	"
9:37	56	95	"
9:38	60	100	"
9:39	60	110	Asleep.
9:40	60	105	"
9:41	60	105	"
9:50	56	90	After rotation in coronal plane
9:51	52	95	Asleep.
9:52	56	100	"
10:10	56	115	After rotation in coronal plane.
10:11	56	115	" "
10:12	56	110	" "
10:13	56	105	" "
10:14	56	115	" "
10:15	56	105	" "
10:16	56	115	" "
10:20	80	90	Resting.

CHAPTER XI

THE EFFECTS OF ROTATION UPON THE DIGESTIVE APPARATUS

A very constant effect of rotations was stasis of the stomach contents. Numerous test meals were given, both with and without drugs, and in no instance was there evidence of even liquids escaping into the intestine. On the other hand, the cardiac sphincter was usually relaxed, for eructations were frequent. In all the rotations performed with the subject sitting upright, i.e., about the long axis, the amount of chyme withdrawn one hour, in some instances two hours, after an Ewald test meal (consisting of two ounces of bread and eight ounces of water), was always some ounces in excess of what had been ingested. In these rotations it was noticeable that the salivary flow was always increased, with marked nausea, and at times a perversion of the sense of smell. In the rotations with the subject lying horizontally, when the rotation was in the sagittal plane the saliva was, in some instances, still somewhat increased, and the chyme withdrawn excessive in amount (e.g., ℥xiv). Nausea and distress referable to the stomach were not a feature in sagittal and coronal rotations, but the stomach was profoundly affected, as shown by the fact that the subject vomited when he left the swing. In horizontal rotations in the coronal plane, and in mild rotations in the sagittal plane, the amount of chyme withdrawn was considerably diminished, compared with that withdrawn after rotations in other planes, and this in spite of the fact that in some instances the K I and salol tests showed the absence of iodin from the saliva, and of salol from the intestine. In these cases it was evident that the diminished amount of chyme was to be attributed to the absence of increased salivary flow, or to diminished secretion from the stomach mucous membrane. On the other hand, in one rotation in the supine posture, and in the coronal plane, there seemed to be a good deal of stomach absorption, because the K I test was positive in 26 minutes, whereas the salol test was negative just after the hour's rotation, and the chyme considerably reduced (℥ iv). Here, evidently, the accretion to the stomach contents was less than the loss by absorption. In these rotations in the supine posture there was no nausea and no increased flow of saliva. It is evident that during the rotations, and some of them were of two hours' duration, nothing passed the pylorus. Rotation is all powerful in keeping the lower opening of the stomach closed. The rotations in the upright (sitting) posture had to be very mild in order that the test meal should be retained. Much less care had to be taken in the rotations in the sagittal plane, whilst in the rotations in the coronal plane, with the subject lying on the back, no amount of rotation could sicken this particular subject's stomach.

The firm and protracted closure of the pylorus in rotations is to be explained by irritation of the vagus centre. If the vagus be divided in the neck, there follows loss of muscle tonus in the stomach, though all contractions do not disappear. Stimulation of the peripheral stump augments the vigour of the contractions of the muscles of the antrum pylori, and causes an increase of tonus in the muscles of the fundus, as well as an increase of the contraction of the pyloric and cardiac sphincters. Occasionally stimulation of the peripheral stump of the cut vagus may cause a primary relaxation or inhibition of short duration of one or all of these structures before the augmentation tonus begins. A similar inhibition can be caused reflexly by stimulation of the central stump of the cut vagus. This effect is absent if the opposite vagus be divided. Hence, the vagus has inhibitor and augmentor fibres, and afferent as well as efferent paths, for the reflex control of the gastric musculature.

Similarly, it can be shown that in the splanchnic nerve there are inhibitor and augmentor fibres for the stomach muscles. Stimulation of the peripheral end of the divided splanchnic causes inhibition of peristalsis and loss of tone. The cells and fibres of Meissner's and Auerbach's plexuses also act as a coördinating mechanism, for the isolated dog's stomach exhibits rhythmic movements for 1 to 1½ hours. The cardiac sphincter remains closed normally, partly from inherent causes, for the closure persists for 24 hours after the severance of all nerves. This sphincter relaxes at the beginning of swallowing, and on stimulation of the glossopharyngeal nerve. Whether the relaxation is due to inhibition of the centre maintaining tonus, or to stimulation of the inhibitory centre, is a question that has not yet been satisfactorily demonstrated. In the isolated pig's stomach, the cardiac and pyloric sphincters remain closed for several hours.

The general contractions of the stomach and intestine are therefore inhibited and augmented by the central nervous system through the splanchnic and vagus nerves. The chief effect of stimulation of the splanchnic being inhibition or diminished tonus, whilst the chief effect of strong vagus stimulation is augmentation of contraction.

Bayliss and Starling[78] consider the rhythmic contractions of the intestines (including the stomach) as myogenic, and propagated from fibre to fibre, and that peristaltic contractions are reflex in character, the coördination being carried out by the local nerve mechanisms initiated by stimulation of the intestine from within. Whether this is so or not, the contractions of the stomach and intestines are augmented and inhibited by the central nervous system through the vagus and splanchnic respectively (Brübaker[79]).

The most plausible explanation of the mechanism of the opening of the pylorus is that based on the action of local coördinating centres stimulating the longitudinal muscle fibres, which, in the region of the pylorus, are collected into thickened bands situated in front and behind, and called the pyloric ligaments, though they contain no white fibrous tissue. These longitudinal muscle strands traverse the pyloric muscle, which is merely a thickening of the circular elements, and pass to the duodenum. It is assumed that in contracting, these longitudinal bands dilate mechanically the circular pyloric ring, being aided by reciprocal inhibition of the pyloric circular fibres. It can be readily seen how augmentation, or failure of inhibition of the circular coat, could overmaster this delicate mechanism, and, as the closed condition is the normal one for the pylorus, the effect of irritation of the vagus centre would be to keep it closed. This closed state of the pylorus and the delicate reflex mechanism by which relaxation occurs are to be considered as defences of the organism against noxious substances, e.g., improperly digested food, etc., reaching the intestine, where they might work serious injury. The resistance of the stomach to all sorts of insult is a matter of daily clinical experience. The stomach has, moreover, in the chemical constitution of its specific secretions, the power to cope with pathogenic organisms. It is therefore considered more in the nature of a reservoir for food rather than as a digestive organ, although there is too much tendency at the present time to underestimate the importance of its digestive functions, no doubt because they are not sufficiently understood. In rotations, the closure of the pylorus, or rather its failure to relax and open, is therefore due to irritation of the vagus centre and to simultaneous inhibition through the splanchnic fibres. It may, perhaps, be considered in the nature of a defensive act on the part of the organism, in part caused, and in part necessitated, by the diminished acidity that usually accompanies it. However, closure of the pylorus (impaired motility) may be present without a deficiency of acid, and frequently occurs in irritated and depressed states of the central nervous system. Most dyspepsias, especially those classed as "nervous," have, like rotation sickness, their origin in fatigue-irritation of the cerebral and medullary mechanisms controlling gastric motility and secretion. The only difference is in the degree and manner of production. We have seen that vomiting in severe states of circulatory depression is frequently a potent means of restoring the blood-supply to the medullary centres. The firm closure of the pylorus, with its accompanying nausea and increased salivary flow, is but a step preparatory in the studied sequence of nature's defences.

Contractions of the stomach, intestines and bladder have been observed by Budge and Valentine [80] to follow irritation of the corpora quadrigemina. These phenomena were regarded by Ferrier [8] as one of the ordinary effects of sensory stimulation. Cannon [81] has shown on cats that, after bilateral splanchnic section, there is no change in the movements of the alimentary canal, whilst after vagus section there were primary twitchings in the starting of gastric peristalsis, with marked weakness of peristaltic contractions, retarded and slow discharge through the pylorus, especially when proteids were fed, and slow passage through the small intestine. Following combined section of the splanchnics and vagi, there were gastric peristaltic waves from the first, causing deep (normal) constriction, which persisted even at autopsy. At first, as in vagus section, there was retarded discharge through the pylorus, with later partial recovery toward the normal when the proteid discharge was more nearly normal than when the vagi alone were cut. Rhythmic segmentation of the intestine was observed in every condition of nerve section. There was rapid discharge of carbohydrate and slow discharge of proteid from the stomach after vagus, splanchnic and combined vago-splanchnic section. Hence the differential discharge of proteids and carbohydrates is confined to a local mechanism.

Auer,[89] working with rabbits, found on bilateral section of the splanchnics that the stomach showed initial weak movements in thirty minutes. Normal peristalsis set in only after two days. The operation was severe, most of the animals dying in a few days. Following subdiaphragmatic section of both vagi, the initial signs of stomach movements appeared in about two hours, and normal peristalsis not until after two days.

Rabbits recover well from this operation, but are likely to develop gastric ulcer at the pre-antral sphincter on the lesser curvature.

After combined section of the splanchnics and vagi, the initial peristalsis appeared in less than thirty minutes. This peristalsis was slow in rate, but almost normal in strength. Peristalsis, normal in rate, rhythm and strength, appeared only after one or two days, and then the gastric waves tended to occur in groups. Rabbits usually recover well from this operation, the mortality being less than after splanchnic, but greater than after vagus section. The rabbits that recover have a greater mortality than normal rabbits, because of reduced general resistance. Only a slight degree of reflex inhibition of the stomach could be got through the vagi, whilst complete reflex inhibition was obtained only when the splanchnics were intact.

After any section, the first signs of peristalsis were seen in the pyloric third. The contractions occurred at normal intervals, but were weak and did not originate in the middle third of the stomach. These first signs of motility occurred during, or at the end of the operation, but normal peristalsis appeared only much later.

That the vagus has some inhibitory fibres had already been shown by Langley,[82] Meltzer,[83] May,[84] and Cannon.[85]

After section of the splanchnics, Auer found it impossible to stop gastric movements by any stimulation which was effective when the splanchnics were intact, with or without vagus section.

This was in corroboration of Cannon and Murphy,[86] who got inhibition of stomach movements through the splanchnics.

Auer's experiments furnish no information for or against the assumption made by Morat,[87] that the splanchnics have a motor function.

It will be noted that Auer saw normal stomach peristalsis only after one or two days following bilateral splanchnic section, whereas Cannon [81 & 88] saw no change from the normal movements. Similarly, after combined vago-splanchnic section Cannon saw normal peristalsis from the first in cats, whilst Auer, in rabbits, saw it only after one or two days. Cannon's animals, after bilateral vagus section, were never so strong as those in which the splanchnics had been cut. Those in which the vagi, or the splanchnics and vagi, had been cut, were asthenic. Auer, however, found that in rabbits, section of the splanchnics was more severe than section of the vagi, or of the vagi and splanchnics.

From these somewhat conflicting results, it is evident that the vagus carries mainly motor influence to the stomach, and that after section of the vagi the discharge of food through the pylorus was slow and retarded. These facts, however, do not warrant the conclusion that stimulation of the vagus would necessarily open the pylorus, or facilitate the passage of food into the duodenum. Many competent observers have found gastric inhibitory fibres in the vagus which may play an important part in the stasis of food content that accompanies rotations. It will be noted that, in spite of this food stasis, the gastric musculature was in a state of hyperactivity during the rotations, as manifested by frequent eructations the "lump-sensation" referred mainly to the pyloric region, and occasional crampy pains referred mainly to the cardiac end. Coincident with the irritation of the vagus centres from rotations, there may have been irritation also of the centres related to the splanchnics. As impulses arriving at the pylorus from these sources tend to counterbalance each other, and as normally, the pylorus is in a state of tonic closure, it would be expected that it should remain closed as the result of rapid rotations. Moreover, the delicate local mechanisms of the pylorus, which seem endowed with almost preternatural powers of discrimination, play an important part in preventing the discharge of poorly prepared gastric contents into the duodenum. In addition, there is diminished hydrochloric acid content, and hydrochloric acid is believed by many to be the immediate exciting stimulus in the normal reflex opening of the pylorus. Afferent impulses through the vagus from the gastric mucosa also play an important rôle. To these we must attribute a large share in the maintenance of pyloric closure during rotations. These afferent vagus impulses undoubtedly are involved in the production of nausea, which, with closure of the pylorus, initiate the sequence of phenomena that culminates in vomiting.

The segments of the cord from which spring the sixth to the ninth thoracic nerves are in relation with the stomach. It is from the cells of the grey matter, especially the central portion, of these segments (spinal vaso-motor centres), that the vaso-motor nerves for the stomach spring. Stimulation of the gastric branches of the vagus has been observed by Rutherford [90] to cause congestion of the gastric mucous membrane, but Burton-Opitz [44] has shown that no vaso-motors for the stomach run in the vagus. The undoubted vascular congestion that accompanies the later stages of rotation sickness seems, therefore, to be due to exhaustion of the vaso-motor mechanisms, causing dilation of the vessels of the splanchnic area, coupled with pallor and extreme contraction of the vessels supplying the surface of the body.

Pawlow[91] has shown experimentally that secretory fibres for the gastric juice are contained in the vagus. He divided the œsophagus in the neck and sutured the divided ends to the skin, thus making two fistulous openings. Later, when the animal was fed, the swallowed food was discharged through the upper fistula without entering the stomach. Pawlow found that such a sham meal caused an abundant flow of gastric juice so long as the vagi were intact, but that no flow was observed when the vagi were cut. Evidently, therefore, the sensation of taste, odour, etc., developed during mastication and swallowing, set up reflexly a stimulation of the secretory fibres in the vagus. Pawlow has called the secretion produced in this way psychic secretion, which implies that the reflex is attended by conscious perception. These experiments further illustrate the close relation between the cerebral motor cortex and gastric function. Psychical secretion, when once started, may continue a long time after the initiating stimulus (e.g., eating) has ceased. This is due to the fact that substances known as secretogogues are contained in the food and in the products of digestion. The action of secretogogues is not as yet completely understood. It is known, however, that they can produce their effect after severance of all the nervous connections of the stomach. Edkins,[92] treating the pyloric mucous membrane as Bayliss and Starling[93] had, similarly treated the mucous membrane of the jejunum, i.e., by boiling, extracting with acids, glycerin, etc., found that decoctions and extracts of the pyloric mucous membrane when injected into the blood caused a marked secretion of gastric juice, whereas mere secretogogues injected into the blood had no such effect. Edkins therefore suggests that secretogogues, whether preformed in the food or formed during digestion, act upon the pyloric mucous membrane and form a substance called gastrin, or gastric secretin, similar to the corresponding secretin for pancreatic secretion, which Bayliss and Starling[93] had demonstrated as being formed in the small intestine. This gastric secretin, after absorption, is carried by the blood to the gastric glands in the middle third of the stomach, and stimulate them to activity by direct action. Starling[94] emphasizes the fact that this mode of control is frequently employed in the body, and suggests the name *hormones* (from *δρμαω*, arouse or excite) for such substances.

Direct stimulation of the peripheral end of the cut vagus causes secretion of the gastric juice only after a long, latent period. The long latency is possibly due to the presence in the vagus of inhibitory fibres for the gastric glands. These fibres being simultaneously stimulated with the secretory fibres, retard the action of the latter.

In rotations, the diminished secretion of gastric juice depends upon a perversion or suppression of all the activities involved in the normal secretion. Thus, the subject knew he was going to be rotated and made uncomfortable, and the meal was not very appetizing (bread and water). These circumstances are very unfavourable for the psychical secretion. Then the meal did not contain preformed secretogogues, as water and bread are poor in these substances, being in marked contrast with meat extracts in this respect. Moreover, the initial effect of vagus irritation being inhibition of secretion, the immediate effect of rotations upon the vagus centre may be assumed to be also inhibition of gastric secretion.

It should also be remembered that the general tendency of strong irritation of the labyrinthine receptors is toward a sickening of the stomach with nausea, mental and physical depression, and vomiting. Such stimulation could hardly favour the secretion of normal gastric juice. The pylorus is also in a state of spasm, which tends to prevent the formation of gastric secretin by the action of whatever secretogogues may have been formed in the progress of digestion.

Finally, owing to impaired absorption, whatever secretin may have been formed in the pyloric or other portion of the mucous membrane, is prevented from gaining access to the glands in the middle third of the stomach. Frequently, however, there was distinct evidence of increased production of fluid in the stomach during rotations. This fluid seems to have been produced mainly in the fundus of the stomach. Time and again, in withdrawing its contents, there was evidence of constriction separating the stomach into two distinct compartments. The contents of each of these compartments frequently presented a marked contrast in appearance and composition. Every clinician must have had similar experience in washing out stomachs. Cannon,[95] and Roux and Balthazard,[98] who studied the stomach movements by means of the X-rays, saw contractions start in the middle region of the stomach and running toward the pylorus. It is possible for these contractions to persist as deep rings seen after death in the excised stomach (Cannon [81]). Hence, in conditions of extreme disturbances of the gastric mechanisms, such as occasionally occur in passing the stomach tube, more especially after rotations, it need not be wondered at if deep constrictions of the circular bands divide the stomach cavity into compartments. Compare the numerous permanent compartments in the stomach of the sheep, etc. [97]

Experiments show that absorption does not readily take place from the stomach. Such substances as water, salts, sugar, dextrins, proteoses, peptones, alcohol, and other drugs, may be absorbed, but not with the same facility as in the intestine. So far as is known, there are no such things as specific nerve fibres directly controlling absorption (Waymouth Reid [268]), yet in some of the rotations there was distinct evidence of increased absorption in instances where the tests showed absolute stasis of the stomach contents. This was notably the case in rotations in dorsal decubitus. However, the possibility of the salol not reaching the intestine with the fluid contents of the stomach must be borne in mind, as well as the possibility that the stomach did not surrender all its contents through the tube.

Salvioli [269] found, notwithstanding the dilated condition of the vessels in a loop of dog's gut following the administration of atropin, that the absolute absorption of peptone and water was less than in a control loop. Waymouth Reid [268] found that less peptone and water was absorbed when the mesenteric nerves were stimulated; after section of these nerves the absolute amount of peptone absorbed was greater, whilst the absolute amount of water absorbed was less than before section. The results following stimulation of the mesenteric nerves are attributed by Reid to the vaso-motor effect. Reid concludes there are no specific nerve fibres for absorption. Techlenberg,[270] however, found marked diminution of absorption rate for K I following section of the mesenteric nerves. As this result is the reverse of what should be expected with the intestinal vessels dilated, Tecklenberg hints at the possibility of absorption being controlled by local mechanisms in the plexuses of Auerbach and Meissner.

Experiments were made to test the effect of hydrochloric acid content upon gastric stasis in the disturbances that attend rotations. On two occasions test meals, consisting of two ounces of bread and seven ounces of water, containing free HCl .182% by weight, were given upon an empty stomach, and mild rotations about the long axis kept up continuously for one hour. The tests showed no absorption and no passage of food into the intestines. In these cases the salol was administered at the beginning of the meal, and in one case the subject had been given hypodermically strychnin sulphate gr $^{1}/_{30}$ twenty minutes before the test meal. The amounts of chyme returned after one hour were ℥ xi and ℥ ix, the latter being from the subject who had had the strychnin. The analyses showed some interesting facts. The specimen from the subject who had had strychnin showed: total acidity .153, free HCl .065, combined HCl .065; whilst that from the other subject showed: total acidity .248, free HCl .153, combined HCl .080. In a control test meal given the latter subject, consisting of the ordinary Ewald meal, and retained for one hour, the figures were: amount of chyme ℥ iii, total acidity .233, free HCl .131, combined HCl .041. As a further means of control, two ounces of the bread used in the meals were soaked in ℥ vii of water containing .182% of HCl. This was kept at a temperature of 100° F. for one hour. It was found that the bread had absorbed the whole of the fluid and become a soddened mass, with a bulk of nine fluid ounces. Fluid was expressed from this mass by squeezing in plain sterilized gauze. This fluid was filtered and analyzed, as usual. The figures were: total acidity .138, free HCl .102, combined HCl .036. The starches and proteins had undergone little, if any, change. In the two test meals given with the acid, the protein digestion seemed to have been carried on well, and the tests for protein enzymes showed that these bodies were present in normal amount. The digestion of the starches seemed retarded somewhat, however, there being marked evidence of free starch, with traces of amylodextrin, whilst there was little or no evidence of erythrodextrin or achroödextrin. There was no maltose, or other reducing agent for Fehling's solution, beyond what was found to have already been present in the control mixture of bread and acid, both immediately after mixing and after being allowed to stand at 100° F. for one hour. The tests for starch digestion in the meals given with the acid presented a marked contrast when compared with the results of similar test meals given without acid. They show conclusively that the presence of acid from the beginning of a meal is a great hindrance to the digestion of carbohydrates. The coagulating enzymes were normal.

These experiments show that the presence of free HCl in the stomach is not sufficient to overcome the resistance offered at the pylorus to the forward propulsion of food during rotations. They also seem to show that the presence of acid in the stomach favors the formation of peptonizing and coagulating enzymes, perhaps by direct stimulation of the gland-bearing area. Moreover, because there was no absorption, and because stasis was complete, the increased amount of chyme, ℥ xi, as compared with the quantity of material ingested, points to the secretion of fluid by the gastric mucous membrane, even making allowance for some increase of saliva which might have been accidentally swallowed.

The fate of the acid put into the stomach seems, to some extent, a problem. In the case where the free HCl was so much reduced, the amount of the chyme was ℥ ix, which was exactly the bulk represented by the meal ingested. Making allowance for the saliva swallowed with the meal, and perhaps for some shreds and detritus from the gastric mucous membrane, the quantitative figures nearly resemble those from the control mixture of bread and acidulated water. It may be noted that this subject never forms free HCl in rotation tests, and the evidence indicated that he did not do so here. It is interesting to note, however, that his coagulating and peptonizing enzymes were normal here, though on several other occasions they were found diminished or absent in rotation tests. There may, however, have been some gastric juice in the stomach before the test meal was given on this occasion, as the stomach was in the resting state, i.e., fasting, in the early morning. This, however, is not at all probable, as on numerous occasions the stomach contents, when removed, whilst the subject was fasting in the early morning, showed little or no evidence of free HCl.

In the other subject there was an increase of free HCl as compared with the figures obtained from the control mixture of bread and acidulated water. This is explained by the fact that with this subject, even in severe rotations, the tests always show the presence of free HCl though this is always diminished in amount, generally about .09% to .10%. Making allowance for dilution by saliva and by gastric secretion, this amount, added to that obtained from the artificial meal (bread and acid mixture), would bring the figure near that obtained from the test meal with the acid.

Another effect of rotation upon the stomach was failure of absorption, which occurred frequently, even when the salivary flow was intense. Diminished acidity was a constant feature, and was present with diminished, as well as with increased, saliva. Hence, the production of stomach acid was retarded. With this, there was found at times absence or diminution of the peptonizing enzyme (pepsin), and even of its zymogen (pepsinogen). Less frequently there was also diminution of the coagulating enzyme (rennet, chymosin), and very rarely of its zymogen (chymosinogen). The tests were numerous and were carefully repeated whenever the enzymes were found to be abnormal. In this way many apparent abnormalities were ruled out. Nevertheless, the facts as above stated were found to obtain. Similarly, in the tests for absorption, where the reaction for iodin in the saliva proved positive, the greatest care was subsequently observed so as to eliminate the possibility of error. As to the excretion or secretion of liquids into the stomach cavity by its mucous membrane, the conclusion is that such a thing occurs during rotations. The increased chyme, however, always seemed to bear some direct relation to the increased saliva. The low acidity and the diminished ferment action, as well as the absence of excessive amounts of mucus, seem to strengthen this view. Moreover, from time to time during the rotation experiments, fairly positive proof was exhibited of congestion of the stomach, as evidenced by hæmatemesis, both spontaneous and on attempting to wash out the stomach. Care was taken to eliminate cases in which the hemorrhage might possibly be attributed to the stomach tube. The congestion of the abdominal viscera harmonizes with what is known to occur on failure of the general circulation due to vaso-dilatation of the splanchnic area. Similar evidence of gastric congestion was apparent, both in aural irrigations and in conditions of sea-sickness. Finally, vomiting of the projectile type forms the climax of the stomach disturbances associated with rotation sickness.

A rational interpretation of the phenomena of nausea and vomiting, as observed in rotation sickness and other disorders, seems impossible unless the existence of a vomiting centre or of some similar mechanism be assumed. The exact location of such a centre may be disregarded, as for present purposes it is immaterial whether it be confined to the medulla, cerebellum or mid-brain. Such a centre would naturally have the most widespread relations with the various receptive areas in different parts of the body. The relations would, of course, be more intimate with certain receptor fields, e.g., the fields of distribution of the senory vagus arcs, and of the labyrinthine and olfactory nerves. The relation with the cerebellum may be only an indirect one, the nausea and vomiting incidental to cerebellar disease being the result of disturbances created in the cerebello-cerebral and cerebro-medullary (efferent) circuits. In other words, the nausea and vomiting in cerebellar disease may result from the vertigo incidental to cerebellar disturbances. The same is probably true for the nausea and vomiting that follow disturbances of the semicircular canals, whilst there is reason for believing that the otolithic apparatus of the vestibule is mainly in direct relation with the nausea and vomiting mechanism. The fact that so many afferent paths play upon a single motor mechanism affords abundant opportunity for mutual reenforcement (*bahnung*) of impulses arriving at the common centre by allied arcs from various receptive areas. The lowering of the threshold at the final common path through irritation in one receptive field may abnormally enhance the value of stimuli acting in other receptive areas. Thus, in rotation sickness, when gastric function has been seriously disordered and nausea has become established, the presence of food or mucus in the stomach becomes an important secondary source of distress until relieved by vomiting. It is probable that the nausea caused by morphin is produced by the effect of the drug, or of its oxidation products, upon the gastric receptors partly, and partly upon the nausea and vomiting centre. The well-known disagreeable subjective complex variously termed "nausea," "the feeling of nausea," etc., has its immediate origin in the cerebral cortex, and is the psychic equivalent of the motor disturbances originating in the vomiting centre.

It should be mentioned that, although the fibre connections between the olfactory paths and the medullary nuclei have not been completely traced, it is highly probable that such connections exist by way of the cortico-mammillary tract from the uncus and hippocampus to the mammillary nucleus, and thence, (1) by way of the mammillo-tegmental tracts proper to the tegmentum of the mid-brain, and possibly to lower levels; (2) by fibres of the pedunculus corporis mammillaris to the tegmentum, which Kölliker, however, believes end in the grey matter about the aqueduct of Sylvius, near the origin of the fourth nerve; (3) possibly by fibres to the posterior longitudinal fasciculus via the hypothalamic commissure. Another and more direct path for impulses from the olfactory receptors in the nasal membrane lies by way of fibres in the strand from the cortex of the olfactory bulb to the thalamus, thence by way of the thalamo-mammillary fibres to the mammillary nucleus, and thence by way of the mammillo-tegmental tracts to the tegmentum and lower levels. For reasons often repeated, it does not seem correct to attribute to any single cranial or spinal nerve nucleus the coördination that is necessitated by a combination of acts involving widely separated structures. Each nucleus, it may be admitted, has its coördinating functions proper to itself, and also acts subordinately to a higher coördinating power, which, by means of its manifold connections with the various lower and higher levels, brings about the harmony of associated action. In fishes there is reason to believe that the mesencephalon is in intimate relation with the labyrinths and with the mechanisms of nausea and vomiting. In the higher forms, with the greater development of the cerebrum and cerebellum, accompanied, in some instances, with recession of the olfactory organs, newer relations have been superadded by which cerebellar and spontaneous psychic impressions exert a profound influence over the motor neurones directly concerned in nausea and vomiting. Labyrinthine impressions are so intimately associated with cerebellar function that whatever influence they have in the causation of nausea and vomiting must come, it seems, mainly by way of the cerebellum, for disturbances of equilibrium always precede nausea and vomiting in rotation sickness. Nevertheless, it is probable that the otolithic apparatus is directly related to the vomiting mechanism. Similarly, retinal impressions which, in the higher forms, are so bound up with cerebral development and psychic function, affect the vomiting mechanism, for the most part, indirectly through the cerebello-cerebral and cerebro-medullary circuits, as well as through sensory psychic pathways.

The perversion of the sense of smell, as evidenced by the painfully disagreeable odour of tobacco-smoke, is another instance of "bahnung," i.e., reënforcement through allied reflexes having reflex arcs, which, though beginning in receptor organs of different species, are nevertheless allied arcs and act harmoniously on the same common path. The absence of vomiting, or the lessened tendency to it observed whilst the subject was in the recumbent posture, and more especially seen in dorsal decubitus, may be accounted for not only by the lessened demands made on the neuro-vascular mechanisms and the greater ease with which the medullary and cerebral circulation is maintained in recumbency, but also to a great extent by the absence of efforts at balancing. These factors combined, reduced the disturbances consequent upon labyrinthine irritation to a minimum. The labyrinthine receptors are acted upon during rotations in recumbency just as efficiently as in rotations with the subject in the erect posture. The impulses generated in them are transmitted centrally along the afferent arcs, but they are incapable of exciting the neurones of the final common path to reflex activity, i.e., the threshold at the commencement of the final common path is too high so long as the subject is in recumbency. When, however, he attempts to stand up, there is a tendency to medullary anæmia, and the demand made upon the neuro-vascular system by the erect posture, coupled with efforts at balancing under the influence of unusual and irregular excitations in the labyrinthine receptors, causes a mutual reënforcement ("bahnung") between the various afferent arcs, with a lowering of the threshold at the commencement of the final common path. If the stimulation of the labyrinthine receptors be of mild degree, only moderate reactions are evoked in the motor neurones of the final common path, or perhaps only a part of the neurones of the final common path are involved, causing slight nausea, salivation, mild contractions of the stomach musculature, the sensation of "lump" in the stomach, or of "lump in the throat." The latter sensation is due probably to abnormal irritability, or perhaps irregular muscular contractions in the upper part of the œsophagus, as evidenced by the frequent voluntary attempts at swallowing made by the subject to overcome the "feeling" and ward off the oncoming sickness. The "lump-sensation" in the stomach is probably due to contraction of the fundic or pyloric musculature. Where the stimulus can be graded, various degrees of reaction may be obtained, from slight disagreeable sensations to the utmost degree of distress, which reaches its climax just before vomiting.

The frequent eructations that occur in rotation sickness are the result of contraction of the gastric musculature, coupled with relaxation of the normal tonic contraction of the cardiac sphincter. The diminished acidity favors eructations, since Cannon [98] has shown that in animals regurgitation readily takes place from a stomach filled with neutral fluid, whereas if the fluid be brought to the degree of normal acidity, no regurgitation takes place. The effect of the acid in preventing regurgitation is seen when both vagi have been cut. Hence, it is to be attributed to local mechanisms.

Rotation sickness was much more readily induced in June than in January and February. Thus, in June a few mild turns of the chair brought the subject to the verge of vomiting, whereas in the colder months, repeated brisk rotations could be withstood by the same subject for quite some time before the onset of nausea. The explanation of this fact rests on the general tonic effects of cold upon the vaso-motor mechanisms, whereby the general and medullary circulation was well maintained and the disorders of equilibration consequent upon unusual or irregular stimulation of the labyrinthine receptors were readily compensated. In direct contrast with these are the general relaxing effects of the summer warmth. The direct effect of cold upon the labyrinthine receptors should also be borne in mind. It is possible, judging from the effects of aural irrigations, and from certain clinical observations, that in cold weather the functional activity of the labyrinthine receptors is depressed, i.e., that the threshold for stimuli is raised in them.

During rotations in various planes there frequently occurred a sense of uneasiness in the bowels, with occasional passage of flatus. In susceptible subjects, after severe rotations, there were general tremulousness and muscular weakness, which persisted in some instances for hours after the rotations, especially in those cases in which vomiting did not occur, and the stomach was not otherwise relieved of its contents.

The conclusions are:

1. That rotation profoundly affects the stomach, and presumably also the intestinal functions, as manifested, (a) by persistent closure of the pylorus, with stasis of the stomach contents and relaxation of the cardiac sphincter; (b) by congestion of the gastric mucous membrane, with diminished absorption; (c) by impaired secretion of gastric juice, with relative and absolute diminution of the specific acid and enzymes; (d) by nausea and increased salivary flow; (e) by general distress and wretchedness, referred especially to the stomach; (f) by eructations, passage of flatus, etc., and vomiting.

2. That these disturbances are initiated primarily by irritation and irregular stimulation of the labyrinthine receptors, which in turn affect the nausea and vomiting mechanism (vomiting centre), directly, perhaps, in the case of the receptors in the maculæ of the vestibule, and indirectly via the cerebello-cerebral and the cerebral-efferent circuits in the case of the receptors in the semicircular canals.

3. That when rotation sickness has been induced, the presence of food, mucus, or other material in the stomach becomes a distinct source of secondary irritation, and tends to maintain and aggravate the condition until relieved by vomiting.

4. That nausea, subjectively considered, is the psychic equivalent of a minor degree of disturbance in the vomiting centre.

5. That gastric, olfactory, visual, and other impressions, with their psychic associations or equivalents, if of sufficient intensity may, of themselves, under ordinary conditions, initiate the phenomena of nausea and vomiting, whilst under conditions of increased irritability of the vomiting centre, from whatever cause, impressions from any of these receptor areas, though of ordinary intensity, may evoke nausea and even vomiting.

6. That the majority of gastric disturbances with their well-known associated sensory phenomena have their origin, primarily at least, in a weak and hyper-irritable condition amounting to a local neurasthenia of the nervous mechanisms, especially those springing from the cerebral cortex which control gastric secretion and motility.

CHAPTER XII

THE EFFECT OF DRUGS AND OTHER MEASURES UPON DERANGEMENTS OF THE ALIMENTARY SYSTEM CAUSED BY ROTATION SICKNESS

The following methods were tested in order to determine their effect in preventing rotation sickness, and especially the gastric and intestinal disorders attending it. The effect of posture was tried by rotating the subject in various planes and noting the result upon the subject's general condition, but more especially upon the gastric motor and digestive functions. For the latter purpose numerous test meals were given upon an empty stomach and retained for one or two hours, during which the subject was rotated constantly. With the subject sitting upright, the rotations about the long axis had to be very mild, as these rotations readily induced vomiting. Rotations in recumbency were borne much better, especially those in the coronal plane, with the subject in dorsal decubitus. Rotations in the sagittal plane, "face forward," with the subject lying upon the side, were also well borne, but rotations in the sagittal plane, "occiput first," were almost as potent in causing subjective distress and gastric disturbances as rotations about the long axis with the subject sitting upright.

The effect of hypnotic suggestion was next tried. Owing to the exaggerated claims made by enthusiasts on behalf of hypnotism as a curative agent in all sorts of conditions, including seasickness, gastric disorders, and even organic disease, this part of the subject seems of sufficient importance to warrant a somewhat free transcription of the notes made during the progress of the experiments.

The subject, a youth of 19, was a fairly normal individual, with normal ear functions. Six swings were given him in the sitting posture about the long axis of the body.

The characteristic after-nystagmus, with its attendant phenomena, were observed. There was dizziness, nausea, inclination of the head, deviation in walking, etc.

The subject was then put into the hypnotic state and placed in the swing. No suggestion was made to him. With a swing from left to right, the subject had to be supported to prevent him falling from the swing. After the swing, he could neither balance well nor walk steadily. Immediately he was taken out of the hypnotic state, and he said he felt wretched and "sick at his stomach." There was no vomiting. Under hypnosis, the suggestion was now made to him that his stomach sickness would disappear, and at once he said it was "all gone now." On being taken out of the hypnotic state, his stomach appeared to be all right. He had no sickness referable to that organ, but he had a headache. Under hypnosis it was suggested that his headache would disappear, which it did immediately. With the subject under "waking" hypnosis, it was suggested that the swing could not sicken him or make him dizzy. A hard swing was then given. Little dizziness was felt, and on being taken from the swing he stood and walked fairly well, but with studied effort and cautiously. He was able to stand fairly well on either foot, with closed eyes. This observation tends to confirm that of Beard.[99] The rotations in Beard's experiments, however, were of a very mild type. The subject now felt slightly sick at the stomach, and the saliva was flowing freely. The knee-jerks were normal. The head felt a little heavy. During a rest, and whilst still under hypnosis, it was found that no suggestion of darkness or of a strong light would alter the size of his pupils.

Still under hypnosis, an attempt was made to introduce the stomach tube, but no amount of suggestion could overcome the rebellious pharyngeal reflexes. It was then suggested to him that he was sick and about to vomit, and at once he vomited. Later he was made to balance on one foot, and whilst in this attitude it was suggested that he was unable to put the other foot to the floor. As he grew tired, desperate efforts at balancing were made, but the other foot was not put down. Finally, the effect of the suggestion was overpowered and the subject put down his foot and walked over to the middle of the room to expectorate.

At 9:30 a. m. an Ewald breakfast was given. At 9:35, after a rotation about the long axis, the usual nystagmus was presented.

During a swing from left to right about the long axis, it was noted that no amount of suggestion could ward off the bad feeling. Instinctively the subject tried to lie down. After a swing in the coronal plane, with the subject in dorsal decubitus, the typical nystagmus was observed. The subject put his hands to the epigastric region as if suffering from stomach distress. He stated he felt sick in his stomach and head. He also had headache and his saliva was flowing freely. His skin felt cold, and he complained of chilliness and headache. The operator laid his hand upon the subject's forehead and assured him that his headache would disappear, which it did at once. There were frequent eructations at various times. The subject manifested a great tendency to lie down and go to sleep. He was allowed to sleep a short time, and on waking he shivered as with cold and complained of headache.

At 10:35 the stomach contents were removed, mainly by vomiting. The amount returned was ℥ xii. Analysis showed a marked diminution of enzymes and of free HCl. No absorption or motility tests were made, but the indications were those of complete stasis and diminished absorption. Before taking him out of the hypnotic state it was suggested that his memory of all the disagreeable incidents of the morning would be blank, and that he would feel well and suffer no discomforting after-effects. On coming out of hypnosis, the subject said: "It's cold. There's something in my mouth that smells." Afterward he told me that his only recollection of what had happened was that he had been dizzy and had felt a choking in his throat. The dizziness was a memory from the swing, whilst the choking was due to the attempts made to introduce the stomach tube. Some time afterward the subject said that he felt the effects of the rotation sickness for a whole week. No amount of persuasion could induce him to permit a repetition of the experiments, though he submitted to numerous irrigation tests. All through the swings, given with the test meal in the stomach, it was constantly suggested to the subject that the swing could not make him dizzy or sick in any way.

Next was studied the effect of extracts made from various portions of the mucous membrane of the stomach and intestines, after the manner followed by Bayliss and Starling,[93] and by Edkins.[92] As these experiments belong to an independent series, the full details are reported elsewhere. The results showed, however, that in rotation sickness these extracts favoured the production of free HCl and digestive enzymes and aided absorption and motility, both in the stomach and intestines.

Finally, various drugs were tested, some singly and some both singly and in different combinations. The following were tried: atropin crystals gr $^1/_{45}$ hypodermatically, ditto *per os;* hyoscyamin gr $^1/_{50}$ hypodermatically; orthoform gr v. *per os;* hyoscin hydrobromid gr $^1/_{100}$ hypodermatically; atropin sulphate gr $^1/_{100}$ *per os;* cocain hydrochlorid gr $^1/_4$ *per os;* atropin sulphate gr $^1/_{75}$, and strychnin nitrate gr $^1/_{40}$ in combination hypodermatically. The drugs were tested by rotating the subject in various planes and postures when a reasonable time had elapsed after their administration, and observing the controlling effects, if any, upon the sickness. With some of the drugs, test meals and controls were given. The test meals were retained for one, in some instances two, hours. During this period the subject was constantly rotated in various planes and postures.

Of the drugs experimented with, none was found to have any appreciable effect in relaxing the pylorus. In this respect, supine recumbency seemed to have the most potency, especially when used in conjunction with atropin and strychnin hypodermatically. In the rotations about the long axis, with the subject sitting upright, atropin and strychnin had no effect in relaxing the pylorus. After atropin, the amount of chyme was considerably increased. In two out of three trials the K I test for absorption was positive. In one case the saliva was diminished distinctly, and in the other two there was no increased salivation. Moreover, nausea was very slight or absent in all three trials. We are therefore driven to the conclusion, since the test for salol in the intestine was negative, that secretion or transudation was responsible for the increased chyme. With strychnin and atropin combined, and with the subject in the supine posture and rotated in the coronal plane, there was active absorption from the stomach, the K I test reacting for iodin in the saliva in 26 minutes, whilst the test for salol in the intestines was negative, and at the same time the chyme was reduced in amount and of a less fluid consistency than usual. Recumbency and the plane of the rotation were the chief factors here, whilst the atropin and strychnin acted as adjuvants. The atropin and strychnin had their usual happy effect in preventing the general discomfort of the subject. Neither cocain nor orthoform *per os* was found to aid absorption or motility, nor was either drug of benefit in preventing dizziness, nausea, excess of saliva, or other discomfort. Hyoscin was ineffective against dizziness, nausea, etc., and caused so much psychic and motor depression that the subject had to be taken home and put to bed. There were no danger signals, however, the chief symptom being an uncontrollable tendency to sleep.

The following figures represent the averages taken from analyses made after Ewald test meals retained during rotations. The general effect of rotation sickness upon the acid content was as follows: The average taken from two Ewald breakfasts, which were retained one hour under normal conditions without rotations, was: total acidity 30%, free HCl .19%, combined HCl .06%.

The average from fourteen meals, retained during rotation in various planes, with the subject in various postures, was: total acidity .17%, free HCl .09%, combined HCl .06%. The average in meals retained during rotations about the long axis was: total acidity .02%, free HCl .10%, combined HCl .07%. The average for four meals after rotations in the coronal plane, with the subject lying in the supine posture, was: total acidity .17%, free HCl .08%, combined HCl .06%.

In rotations in the sagittal plane, with the subject lying on the side, the average for three meals was: total acidity .12%, free HCl .06%, combined HCl .03%. In rotations about the long axis, with the subject sitting upright, and after the administration of atropin, the average for four meals was: total acidity .20%, free HCl .11%, combined HCl .08%. In rotations about the long axis, with the subject sitting upright, the rotations and reverses had to be of the mildest kind to forestall vomiting, whereas in rotations with the subject lying down there was little tendency to vomit, even when the rotations were brisk and the reversals or stoppings rather sudden.

In rotations in the coronal plane, with the subject lying on the back, and after the administration of atropin and strychnin, the figures were: total acidity .18%, free HCl .07%, combined HCl .08%.

The lowest figures occurred with rotations in the sagittal plane, including rotations with "face first" and with "occiput first," e.g., total acidity, .07%, free HCl .029%, combined HCl .029%. The next lowest were with rotations in the coronal plane, with the subject in hypnosis, and under the suggestion that the rotation would not sicken him, etc. The figures were: total acidity .12%, free HCl .03%, combined HCl .08%. There was a trace of blood in the chyme.

Rotation in the coronal plane, with the subject in the prone posture, gave: total acidity .18%, free HCl .08%, combined HCl .05%.

The contents from the resting stomach gave, as an average: total acidity .14%, free HCl .06%, combined HCl .05%.

The amount of chyme withdrawn after one hour's retention during rotations was as follows: Average after five meals in rotations about the long axis, ℥ xiiss; after four meals in rotations in the coronal plane, with the subject lying on the back, ℥ x; after sagittal rotations, with the subject lying on the side, for three meals the average was ℥ xii; after rotations about the long axis, with the subject sitting upright, and after the administration of atropin, the average for four meals was ℥ xii. After rotations in the coronal plane, with the subject lying on the back, and after the administration of strychnin and atropin, the amount for one meal was ℥ ix. The general average for all the meals under rotations in the various planes and postures was ℥ x. In each instance the meal consisted of, bread ℥ ii and water ℥ viii.

The peptic ferments were found diminished or absent in the three cases where the acidity was very low, viz., in rotations in the sagittal plane, in the coronal plane with the subject lying prone, and in the coronal plane with the subject under hypnosis.

The salivary flow was markedly increased in rotations about the long axis, with the subject sitting upright. It was increased slightly, or not at all, in rotations in the sagittal plane, with the subject lying on the side. In rotations in the coronal plane, with the subject lying on the back, it was diminished or not affected. After atropin it was always considerably diminished.

Starch digestion seemed to have been well carried on in every instance, aided, no doubt, by the lowered acidity and increased salivation. The tests for motility showed that in no instance was there evidence of anything having passed the pylorus.

The absorption tests showed that in very few instances was there convincing evidence of absorption from the stomach. In two of these cases the rotation was in the coronal plane, with the subject lying on the back, and in one of these rotations the subject had had atropin and strychnin. In two rotations about the long axis, with the subject sitting, upright, and after the administration of atropin, the K I test reacted for iodin in the saliva, but in these cases there was reason to believe that contamination of the saliva had taken place on the administration of the K I, as some of the drug was found loose in the box containing the capsules. In subsequent tests special means were adopted to avoid such an accident. The remaining tests showed that no absorption took place from the stomach, and this, even allowing for the diminished salivary secretion, which was never extreme.

Atropin, especially in combination with strychnin, afforded almost complete relief from stomach discomfort, nausea and increased salivary flow. Vertigo was present, however, during the rotations, but on cessation of the latter it immediately disappeared, and was not accompanied by the usual dolor cerebri. The subject had not the usual fear and apprehension of the swing. Instead of appearing worried and morose, he was cheerful, talkative, and anxious to work. In a word, his manner and bearing gave evidence of the usual atropin action on the cerebral psychic and motor areas. Atropin manifestly exerts a powerful local action on the gastric vagus sensory terminals. This local action, to the infinite relief of the subject, eliminates the stomach and its contents as powerful secondary factors in maintaining vagus nuclear irritation. In other respects, the atropin showed its usual effect in stimulating the cerebral (psychic and motor) areas and the medullary centres. Its action on the latter has been discussed elsewhere.

The general effect of atropin *per os* was good, as was that of hyoscyamin hypodermatically. However, neither was as efficient as atropin or its combination with strychnin hypodermatically.

Experience with other drugs in making the experiments to determine their effect upon the circulation in rotation sickness rendered it inadvisable to try their effect upon the digestive apparatus. The results upon the circulation and upon the subject's condition generally were so unsatisfactory that nothing good could be expected from their action upon gastric or intestinal function.

The conclusions are:

1. That dorsal decubitus is the most efficient method of counteracting the nausea and distress in rotation sickness.

2. That atropin and strychnin in combination favour dorsal decubitus, aiding its action in tending to relax the pyloric sphincter, whilst, without decubitus, these drugs as well as cocain and the other drugs tried, are ineffectual.

3. That during rotations in various planes, and with the subject in various postures, there is active secretion or transudation from the gastric mucosa into the stomach cavity, even when the system is under the influence of atropin.

4. That this secretion or transudation represents a gastric juice of lowered acidity, and in some instances of diminished or absent enzymes.

5. That the erect posture has much to do with the induction of nausea, salivation, and vomiting, whilst the recumbent posture, and especially dorsal decubitus, prevents or alleviates them.

6. That hypnotic suggestion is not markedly effective in offsetting the effects of rotation sickness, nor in preventing the usual disturbances in gastric function and their immediate attendants.

7. That atropin, especially in combination with strych-

nin, is very effective in combating the nausea, increased salivation and vomiting, and the psychical and mental depression that accompany rotation sickness, and that it does this, (1) by local action on the sensory vagus terminals in the gastric mucous membrane, thereby preventing secondary irritation of the vagus and allied centres, and, (2) by stimulating the psychic and motor areas of the brain, as well as some of the medullary centres, e.g., vaso-constrictor, respiratory, etc. The usual potency of the disturbed labyrinthine and gastric receptors is, to a great extent, offset or prevented by this twofold action of atropin. The strychnin acts as an adjuvant in maintaining the circulatory and general tonus.

8. That atropin *per os* and hyoscyamin hypodermatically are effective in counteracting some of the effects of rotation sickness, but not to the same degree as atropin, or the combination of atropin and strychnin, used hypodermatically.

9. That cocain and orthoform *per os* are ineffective, whilst hyoscin hypodermatically is not only ineffective, but seems to aggravate the phenomena evoked by rotation sickness.

10. That certain extracts made from various parts of the mucous membrane of the stomach are effective in rotation sickness, and materially favour absorption and motility in the stomach and intestine.

CHAPTER XIII

THE EFFECT OF ROTATION UPON EQUILIBRIUM

In rotations about the long axis, with the subject sitting upright, rotation from right to left (i.e., in the direction opposite to that of the hands of a watch, supposing the watch to be placed on the floor immediately under the swing and with its face looking upward, and the axis of its hands parallel to the long axis of the subject's body), causes the subject's head to incline toward the right shoulder. If the swing be allowed to slow down gradually, a stage is reached when the subject does not know to which side his head tends to fall, as it seems equally inclined to fall laterally to the right or left. This stage, if the swing be allowed to slow down still further, is soon followed by a period when there is a distinct tendency of the head to fall to the opposite, i.e., to the left shoulder. In swings from left to right, similar phenomena occur, but in this instance the initial inclination of the head is toward the left shoulder. There is also at the beginning of the rotation some rigidity and inclination of the upper part of the body in the same direction in which the head inclines. Sudden acceleration, retardation, or reversal of the direction of the rotation, has a powerful influence upon these deviations of the head, as well as upon the vertigo and other phenomena attending rotation. If the eyes of the subject be kept closed and a brisk rotation performed from right to left, and the swing allowed to slow down, the subject's head will at first tend to fall to the right, and he will feel himself rotating from right to left. Very soon, however, as the swing slows down, his head will not tend to fall in any particular direction, and the subject will feel no sense of motion whatever. As the swing still continues to slow down, the head begins to incline slightly to the left and the subject now feels himself revolving from left to right, though in reality he is still revolving from right to left. At any time during such a period as that just described an acceleration of the rotation will determine the head to fall in the direction opposite to that of the acceleration, and the subject will feel himself rotating in the direction in which he is actually moving. Similarly, if, after a few short, brisk rotations from right to left with eyes closed, the swing be suddenly retarded or stopped, the subject's head will immediately fall to the left, and he will feel his body rotating rapidly from left to right. If, now, the eyes be immediately opened, the subject no longer feels his body moving, but external objects appear to be moving rapidly about his body from left to right. By opening his eyes, his sense of motion has become externalized, that is, external objects now seem to move, whilst his body seems to be stationary. If the subject be taken from the swing at this point he will be able to walk, but will tend to deviate toward the side to which his head inclines. However, it frequently happens that in a moment or so the head may incline to the opposite side, and the subject will accordingly deviate in that direction.

Observation of the displacement of the head and upper part of the body in rotations in the coronal plane, i.e., about an antero-posterior axis through the umbilicus, was beset with some difficulty, for in these rotations the subject was lying on the back, i.e., in the position of most stable equilibrium. Another element that added to the difficulty was retardation of the swing, which, of course, reverses the direction of the nystagmus, etc. Hence, the notes taken at the time the experiments were made show some irregularities and apparent contradictions. The swings, however, were repeated again and again, with every possible attention as to slowing and acceleration, until the true facts were ascertained as nearly as possible. It may be said in general that rotation in the coronal plane in the direction of the hands of the watch (which in all these experiments with the subject lying down is supposed to be placed on the subject's breast, with its face looking toward the observer), causes a tendency to displacement of the head and upper part of the body toward the right, i.e., in the direction against the hands of the watch. Similarly, rotation against the watch caused a tendency of the upper part of the body to "pull" or incline in the opposite direction.

In determining these matters, it was found that by placing the hands on either side of the neck one could perceive on which side the reflex tonus was exerted in the muscles. In addition to this, the subject experienced a painful sense of effort in the opponents on the opposite side. This was due to relative absence of labyrinthine tonus in these muscles.

The author, who is very susceptible to the effects of rotation, went on the swing himself to clear up this matter. In addition to the facts as stated above, it was found that in rotations in the mesial plane, "face front," little, if any, reflex tonus could be detected, whilst in mesial rotations with the "occiput first" there was, at the commencement of the rotations, a distinct tonus effect detected in the muscles that pull the head forward, whilst in sudden stopping there was marked reflex tonus in the muscles that pull the head backward. The latter was accompanied by a sense of motion in the mesial plane, "face first." This observation, taken in conjunction with the phenomena observed on section of the posterior canals in animals, viz., falling backward, seems to show, as Ferrier[8] long ago pointed out, that mesial rotations with the "occiput first" stimulates the receptors in the ampullæ of the posterior canals. The absence of marked phenomena in mesial rotations, "face first," seems to point to the fact that in forward movements and rotations the eyes have so long and so effectively replaced the related labyrinthine receptors, viz., those of the superior canal, that the latter perhaps have regressed in function and irritability.

With the eyes closed and directed straight ahead, i.e., in the sagittal plane, it was found that in rotation in the coronal plane the subject felt no movement but an up-and-down motion, as if the body "seesawed" in the sagittal plane, with the head, however, always appearing to the subject to be lower than the feet. The possibility of lateral swaying of the swing should be remembered in connection with the phenomena just noted.

When the subject was rotated in the direction of the hands of the watch, but with the eyes closed and strongly deviated to the left, i.e., in the direction in which he was moving, he correctly estimated at the beginning of the rotation the direction in which his body was moving, but with, at times, an added element, viz., the sensation that his body was rotating about its long axis from left to right. The explanation of this latter phenomenon seems to rest upon the fact that superior oblique muscles are attached to the eyeball behind the centre of rotation (Fuchs[100]). The consequence is, that in the production of rotary nystagmus with the watch there is also a tendency to cause movements of horizontal nystagmus, with the short elements directed to the right. This nystagmus, on account of the insertion of the superior oblique behind the centre of rotation of the eyeball, is favoured in the right eye at least, by turning the eyes to the left. Moreover, it is the primary associated equivalent of rotation about the long axis from left to right.

In rotations in the coronal plane with the watch, if the eyes were kept closed and strongly turned to the right, the subject felt, soon after the commencement of rotation and on slight slowing perhaps, his body rotating in the coronal plane against the watch. Here, looking to the right inhibited rotary nystagmus with the watch, and favoured the premature induction of the natural after-nystagmus, viz., against the watch, which is the associated equivalent of rotation in a similar direction.

Another interesting fact in rotations in the coronal plane with the watch, and with the eyes closed and directed to the right, was, that in addition to the sense of rotation against the watch the subject also was aware, from light impressions received through the closed eyelids, that things went "shooting in front of him from the feet toward the head" in the mesial plane, which, of course, is associated with rotation in the mesial plane in the direction "face forward."

If brisk rotation be commenced in the coronal plane with the eyes closed and turned strongly in the direction of the rotation, even then, whilst the subject correctly estimates the actual direction of the rotation, turning the eyes to the opposite side immediately begets a sense of reversal of the motion. Similar phenomena occur with aural irrigations. As already noted, the turning of the eyes favoured the premature induction of the natural after-nystagmus.

In these rotations in the coronal plane, the usual effects of acceleration and retardation, as noted in the rotations about the long axis, are encountered. There are periods when the direction of the motion is correctly estimated, and there are periods when no motion whatever is felt. These latter occur in certain positions of the eyes, as well as just previous to the sensation of reversals of direction which are evoked by gradual retardation.

In rotations in the sagittal, or in a diagonal plane midway between the sagittal and the coronal, the direction in which the closed eyes were turned had a peculiar effect. In studying this particular point, difficulties were encountered owing to retardation effects. However, the rotations were repeated again and again, and from a careful study of the results this conclusion was reached, viz., that in rotation in the sagittal and diagonal planes, with the subject lying on the left side, turning the closed eyes strongly to the right or left, had a strong determining influence on the subject's judgment as to the direction in which his body actually moved or seemed to move, so that if the objective determining influence were only of slight degree, whether this were in the form of active acceleration or reactionary retardation, the voluntary straining of the eyes toward the right or left determined the sensation of motion in the direction of "occiput first" or "face first," respectively, or enhanced the action of the objective influences tending to show their effects by causing a feeling of rotation in either of these respective directions. Thus in rotations with closed eyes directed to the left, there was a marked tendency for the subject to experience a sense of rotation in the direction of "face forward," whilst with the eyes turned to the right the tendency was to evoke a sensation of rotation with "occiput first," and this, regardless of the actual direction of the rotation where there was no sudden or marked acceleration or slowing. These phenomena can be explained just as the similar phenomena noted in rotations in the coronal plane. Thus, turning the eyes to the right, by which they were, in this individual, drawn upward, inhibited vertical nystagmus directed toward the feet, which is associated with sagittal rotations "face first," whilst it favoured vertical nystagmus toward the top of the head, which is associated with sagittal rotations with "occiput first." Similarly, on looking to the left, the eyes were directed toward the feet, which inhibited vertical nystagmus toward the head and favoured it toward the feet, thereby evoking the sensation of rotation "face first."

It would seem that in every form of nystagmus due to rotation there is, apart from the secondary phenomena, a tendency to a superposed subordinate mild nystagmus in the direction opposite to that of the primary nystagmus. This subordinate nystagmus may be so enhanced by turning the eyes in the direction that favours it and suppresses nystagmus in the opposite direction, as to completely overshadow the latter in its effects. The bilateral relation of the peripheral vestibular apparatus to the cerebellum may account for this twofold nystagmus, the more extensive relation predominating in its effects under ordinary circumstances, whilst under the exceptional circumstances mentioned the nystagmus originated under the influence of the lesser relation is permitted to predominate. It is a matter of ordinary experience, however, that strongly turning the eyes in any one direction tends to develop nystagmus in that direction. This seems to indicate that the overlapping functions of the ocular muscles is in great part responsible for subsidiary latent forms of nystagmus.

CHAPTER XIV

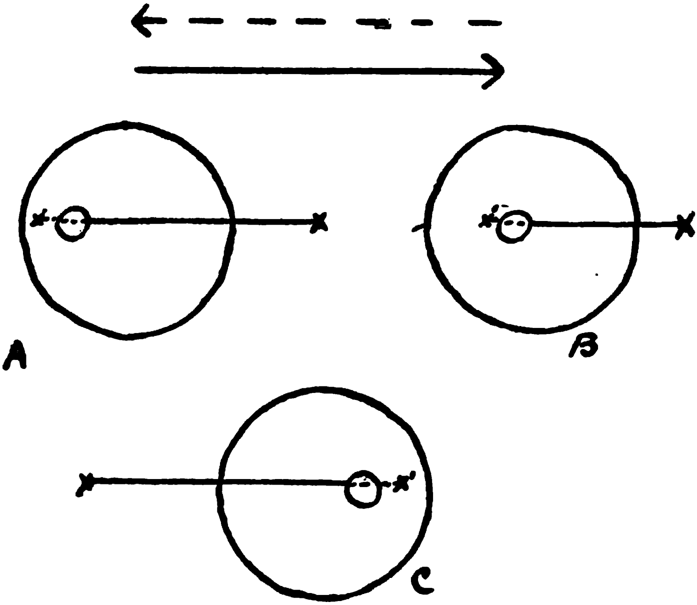
THE EFFECTS OF ROTATION UPON THE EYES

During rotations, certain movements of the eyeballs occur which are nystagmic in character, but which bear a constant relation to the plane and direction of the rotation. These movements of the eyes have been called nystagmus, though there is nothing about them suggestive of the sleepy noddings which the word implies. On the contrary, they are an exaggeration of finely coördinated reflex movements intended to aid visual fixation during the progress of the body, through space, or whilst objects move rapidly past the body, the latter being stationary. A familiar instance of their occurrence under ordinary circumstances is the well-known behaviour of an individual's eyes as he watches near-by objects from a rapidly moving train. The eyes in a series of rapid jerks, during which vision is impossible, jump in the direction in which the subject's body is moving, and then, fixing on the object to be seen, follow it with a movement in the opposite direction. The latter movement has just enough speed to keep the image of the object in a fixed position upon the retina, or as near to it as possible. Vision is, of course, possible during this second or slow movement, for the aim and object of both movements is to render vision as distinct as possible. Although distinctly a misnomer etymologically, the term nystagmus, out of deference to ancient usage, is retained.

In speaking of this nystagmus, the direction of it is in every instance to be understood as meaning the direction of the short movements during which vision is impossible. During a rotation from right to left about the vertical axis, i.e., in the horizontal plane, with the subject sitting upright, nystagmus occurs in the horizontal plane and in the direction in which the body is moving, i.e., toward the left. If the swing be suddenly interrupted or abruptly slowed, it will be found that horizontal nystagmus is present, but directed toward the right, i.e., in the reverse direction of that which it had during the rotation. If the eyes be kept closed during the progress of the swing, nevertheless, they will be found to be in nystagmus directed toward the left, and on interruption or slowing of the swing, with the eyes still closed, the subject will experience a sense of movement in the opposite direction to that in which he may still be actually moving, and at the same time the direction of the nystagmus shifts and is now directed to the right, that is, in the same direction in which the subject believes his body to be rotating. If, at this moment, the eyes be opened, all subjective sense of movement of the body ceases, but external objects appear to be moving from left to right. Some authors have termed this latter phenomenon the externalization of vertigo. The explanation is very simple. During the excursion of the eyeball in the direction of the nystagmus—in this instance from left to right—vision is impossible, but in the return movement of the eye from right to left the images of external objects, seen after the commencement of the slow movement, fall upon successive areas of the retina which are located farther and farther toward the left of the fundus as the eye rotates to the left. This shifting of the images of objects along the retina from right to left is plausibly mistaken for an actual movement of external objects from left to right, and as vision is impossible during the rapid movements of the eye from left to right, the result is an apparent continuous movement of objects from left to right. (See diagrams.)

In very rapid rotations about the long axis, more especially if the subject lean backward so that there is a coronal element in the rotation, the nystagmus may not be of the purely horizontal type, but may be associated with a rotary element about the antero-posterior axis of the eyeball. The direction of this nystagmus in rotations from right to left is primarily, i.e., during the actual rotation before slowing has taken place, in the direction of the hands of the watch (with the latter so placed against the breast of the subject that its face looks toward the observer), whilst secondarily, i.e., on sudden stopping or slowing of the swing, the direction is against the hands of the watch.

In the presence of mixed nystagmus, external objects in general appear to move in the direction of the horizontal element. The apparent movement imparted to them by the rotary element of the nystagmus varies, depending on which side of the central sagittal meridian of the eye the objects are seen from, i.e., whether from the right or left of the individual.



Diagrams to illustrate the apparent movement of objects in horizontal nystagmus. See text for explanation.

In the diagrams, to illustrate the apparent movement of objects in horizontal after-nystagmus, the dotted arrow shows the direction of the nystagmus. The image, X, in the slow return movement of the eye, falls upon successive areas of retina placed farther and farther to the left of the fundus on account of the rotation of the eyeball from right to left about its vertical axis. This rotation causes the anterior half of the eyeball to rotate toward the left, and the posterior half to rotate toward the right, with the result that the image on the retina appears to move toward the left. This is interpreted as a movement of external objects toward the right. Figure A shows X′, the location on the retina of the image of X, at the commencement of the slow return movement of the eye from right to left. Figure B shows the location of the same image a moment later. Figure C shows the location on the retina of the image X′ at the end of the slow return movement, just before the commencement of the rapid series of jerks to the right. During the progress of the slow movement, the degree of apparent displacement of the retinal image X′ corresponds with what would occur in an actual movement of the external object X from the left to the right of the subject if the eyes were stationary.

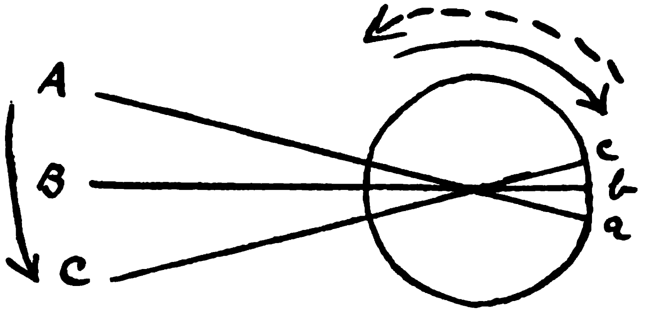


Diagram to illustrate the apparent movement of objects in rotary nystagmus. For explanation see text.

In the diagram illustrating the apparent movement of objects in rotary nystagmus, the dotted arrow represents the direction of the short, rapid element, whilst the continuous arrow represents the direction of the slow element. An object at A to the right of the subject casts its image at *a* on the left side of the retina. As the fundus rotates slowly with the watch, the image at *a* soon falls upon successively higher areas of the retina, represented by *b* and *c* of the diagram. This is falsely interpreted (projected) as a movement of the external object from A to B and C, respectively, i.e., as a movement from a higher to a lower position when the object is seen from the right of the individual.

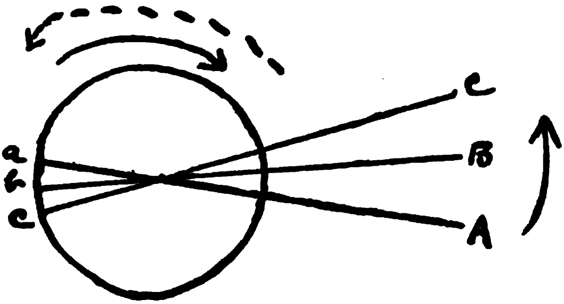


Diagram to illustrate the apparent movement of objects in rotary nystagmus.

In the second diagram, illustrating the apparent movement of objects in rotary nystagmus, an object at A to the left of the subject casts its image at *a* on the right side of the fundus. Owing to the slow rotation of the fundus with the watch, the image at *a* soon falls on successively lower areas of the retina, represented by *b* and *c* in the diagram. This is falsely interpreted (projected) as a movement upward of the object at A to the points B and C, respectively. Thus, because the slow element of the rotary nystagmus is with the hands of the watch, objects seen from the left of the subject seem to ascend, because their images fall upon successively *lower* areas on the right half of the fundus. The moment, however, that the eye, in its horizontal movement, reaches a position in which the objects seen are situated on the right of the central sagittal meridian, so that their images fall upon the left half of the retina, then because the slow rotary element is with the hands of the watch the images appear upon successively higher areas of the retina, and external objects, now seen from the right of the individual, seem to move downward. These facts, which are in full accord with the principles of optics, should be constantly borne in mind, as they afford a satisfactory explanation of the apparent contradictions of necessity encountered wherever the findings in the study of rotary nystagmus are recorded.

In rotations in the coronal plane, the dominant type of nystagmus is the rotary with a horizontal element admixed. In rotations with the hands of the watch, the rotary element is directed primarily, i.e., during the actual rotation and before slowing has occurred, in the direction of the hands of the watch, the horizontal element being directed toward the left; whilst secondarily, i.e., on slowing or sudden stopping of the rotation, the rotary element is directed against the watch and the horizontal element toward the right. During the secondary nystagmus, external objects appear to move around the subject's body in the coronal plane, and in the direction against the hands of the watch, that is, upward on the left side over the head and downward on the right. In the study of rotary nystagmus, however, many irregularities are encountered, since the horizontal element is frequently, in this form of nystagmus, the result, not of coördinated action, but of the peculiarities in the arrangement of the muscles involved. Owing to the insertion of the superior and inferior oblique behind the centre of rotation of the eyeball (Fuchs[100]), there is, in rotary nystagmus, a tendency to the production of a horizontal element of different direction in either eye. Thus, in rotary nystagmus against the watch the short elements of the nystagmus result, in the right eye, mainly from short, rapid contractions in the inferior oblique of that eye, whilst in the left eye, the similar movements are to be attributed to rapid contractions in the superior oblique of that eye. These contractions are responsible for the short elements of the rotary nystagmus against the watch. The secondary effects of the contractions, however, cause a tendency to produce short horizontal movements toward the right in the right eye, and toward the left in the left eye. Such irregularities of function of the muscular apparatus of the eyeball are ordinarily compensated by the action of the other ocular muscles. Strongly turning the eye from one side to the other, tends to develop the irregularity in one eye and to suppress it, perhaps, in the other. It is owing to the development or suppression of various forms of nystagmus that, by strongly turning the closed eyes in one direction or another, the subject can influence the subjective sense of motion he experiences during or after rotations.

Since in rotations these movements occur with the eyes closed, and because various physiologists have observed nystagmus on direct stimulation of the semicircular canals, it is fair to conclude that in rotations nystagmus is mainly the result of irritation of certain labyrinthine receptors, depending upon the form of nystagmus produced. The labyrinthine nystagmus caused reflexly when the body is rotated, is a physiological process intended to facilitate visual fixation, and is associated with other reflex compensatory movements (synkineses), also of labyrinthine origin. However, where the body is stationary and an attempt is made at visual fixation of rapidly passing objects, a visual nystagmus is evoked through the retinal receptors. It is probable that in rotations with the eyes open, the nystagmus is the result of impulses generated in both retinal and labyrinthine receptors impinging jointly upon the same final common paths.

In rotations in the sagittal plane, nystagmus of the vertical type occurs. Rotation in the sagittal plane with the "face first" caused vertical nystagmus, which was primarily directed toward the feet, and secondarily, i.e., after slowing or stopping of the swing, toward the top of the head. External objects "just wobbled," with a general tendency of moving from the feet toward the head. It was a noticeable feature in rotations in the sagittal plane, with the face forward, that the nystagmus was never so intense as that seen in rotations in the same plane with "occiput first," nor had the rotations the same tendency to distress and sicken the subject. Rotations in the sagittal plane with the "occiput first" caused furious vertical nystagmus, directed primarily toward the top of the head, and secondarily, toward the feet. During the after-nystagmus external objects seemed to move rapidly and steadily toward the feet from the head in the sagittal plane.

In rotations about a diagonal transverse axis at the level of the umbilicus, i.e., in a plane midway between the coronal and sagittal, with the subject lying partly on the left side and partly on the back, the chief type of nystagmus was vertical in direction, but with a rotary element added. Rotations in this plane with "occiput first" caused, primarily, vertical nystagmus toward the top of the head, with a rotary element against the watch. The after-nystagmus was toward the feet, with a rotary element directed with the watch. External objects seemed to move from the head to the feet, with a slight diagonal deviation toward the left foot. At times they seemed to move over the head from right to left, with the watch in a plane between the coronal and the sagittal. During the rotation, on looking strongly toward the right, objects flew past the face from right to left. During the "after-nystagmus," on turning the eyes strongly to the left, external objects seemed to move about the body in the direction of the hands of the watch, and at the same time showed a distinct rotary nystagmus with the watch. This latter was inhibited by looking toward the right. On another occasion during the after-nystagmus, objects seemed to the subject to travel in two directions, viz., (1) about his body, partly in the coronal plane and with the watch, and, (2) from right to left about the long axis of the subject's body, whereby the "wall seemed to be sinking into the ground." The latter movement is accounted for by a horizontal element in the nystagmus directed toward the left, which at times accompanies rotary nystagmus, and in certain positions of the eyes seems to dominate it.

In rotations in the diagonal plane, "face first," the secondary nystagmus tends to be mild and of short duration, and is directed chiefly toward the top of the head.

It is noteworthy that the nystagmic movements of the eyes that accompany rotation, i.e., acceleration and retardation, are wholly reflex in their nature. Another noteworthy fact is, that the nystagmic movements during actual rotation always bear a characteristic fixed relation to the direction in which the body is actually moving, whilst during the "after-nystagmus" the direction of the movements of the eyeballs have a characteristic fixed relation to the subjective sense of motion which the individual with closed eyes experiences. The direction of this subjective sense of motion is always with the short elements, just as the short elements of the primary nystagmus are always of the same direction as that of the actual rotation. These two facts, fundamental in importance, suffice for present purposes. The fuller discussion of the subject will receive attention under aural irrigations where the intricate movements of the eyes were studied with the aid of the ophthalmoscope.

The changes that occur in the fundus oculi during rotations are mainly those dependent upon altered conditions of the blood-vessels. Just after a violent rotation the arteries were generally found contracted and practically empty. This condition was soon followed by dilatation, and more or less congestion. Like nystagmus, this part of the subject, as well as the behaviour of the pupils, is best studied during aural irrigations.

In rotations, turning the eyes constantly from one side to the other and concentrating the attention, had a powerful effect in upsetting the general organism and causing distress. This was most noticeable in a series of rotations in the coronal plane, with the subject lying supine, where, under ordinary circumstances, we might expect the least effect upon gastric motility, nausea, etc.

CHAPTER XV

THE GENERAL EFFECTS OF AURAL IRRIGATIONS

The effects of irrigations of the external auditory canal resemble, in general, those of rotations. Disturbances of equilibrium, nystagmus, circulatory changes, nausea, increased salivation, sweating, pallor, vomiting, and general prostration similar in every particular to the phenomena that accompany rotations, occur when water of a certain temperature is allowed to flow continuously for two or three minutes into the external auditory canal. New and interesting features, however, are added, as, for instance, the deviation of the head toward the left with horizontal nystagmus to the right on irrigating the left ear with cold water, whilst irrigation of the same ear with hot water produced similar phenomena but in the opposite direction, that is, the head inclined toward the right, whilst the nystagmus was directed toward the left.

In aural irrigations the temperature of the irrigating fluid is all-important. In this connection, it is to be remembered that the thermic effect of baths depends upon the difference between the temperature of the water used and that of the surface of the body upon which it is applied. It also depends upon the extent of surface to which the water is applied, as well as upon the suddenness and force of impact. As the temperature of the human skin ranges between 90° and 95° F., with an average, perhaps, of 92.5° F., hydriatrists consider a bath of 93° F. or thereabout as neutral, i.e., neither hot nor cold. The same rules regarding temperature, etc., govern aural irrigation, but owing to anatomical and physiological peculiarities the thermic neutral point is some degrees higher for the labyrinth than that for the general skin surface. Generally speaking, aural irrigations at 80° F. produce effects which could not be duplicated by hot irrigations below 110° F., or, we might say, perhaps with more accuracy below 113° F. This would bring the neutral point for thermal stimulation of the labyrinth close to the internal body temperature.

The force under which the fluid is driven into the canal has a certain effect in aural irrigations, but the temperature of the fluid is the chief determining factor in eliciting the characteristic phenomena. When not otherwise mentioned, the height of the irrigating receptacle in the experiments is to be understood as being at 1¾ feet above the external auditory meatus in irrigations with the subject sitting erect, and 2½ feet above the meatus in irrigations with the subject in the supine posture.

Standing erect during the progress of the irrigations has a most powerful influence in aggravating some of the phenomena and sickening the subject. Sitting seems to retard some of the disagreeable symptoms, whilst lying supine has the greatest effect in retarding them, especially the nausea and vomiting, although if the irrigations be prolonged or severe, projectile vomiting may result.

Concentration of attention, as well as muscular efforts at balancing, are important secondary factors in determining sickness and prostration. It was noted in irrigations with the subject lying supine that constantly turning the closed eyes from one side to the other and concentrating the attention to ascertain the direction in which the subject believed his body to be turning, very quickly brought the sickness to a climax, thereby corroborating the findings in rotations under similar conditions of posture.

Within limits of moderate range, bilateral irrigations with water at the same temperature have less tendency to sicken and disturb the subject's equilibrium than unilateral irrigation with water at the same temperature. With very cold or very hot bilateral irrigations, however, intense vomiting can occur, though even with these, the nystagmus seemed to be moderate, or even absent. Thus, in a bilateral irrigation at 67° and in another at 57° F. vomiting occurred, but there was no distinct nystagmus beyond a slight jerkiness, the direction of which was doubtful, but which seemed to be toward the top of the head. There was no apparent movement of external objects. In bilateral irrigations, therefore, the vomiting seems to be due to disturbances in the receptors in the maculæ of the vestibule, which, as before stated, are probably in direct relation with the nausea and vomiting mechanisms. The receptors in the maculæ on either side of the body, in so far as they directly affect the mechanisms of nausea and vomiting, do not seem, therefore, to stand toward each other in the relation of balanced mechanisms as do the ampullary receptors. The macular receptors, however, on either side of the body are set up against each other as balanced mechanisms, in so far as they affect other mechanisms, e.g., those of equilibration and possibly those of nystagmus.

The Effects of Aural Irrigations upon the Circulation

The chief effect of cold or hot irrigations is an immediate rise in blood-pressure, with a slowing of the pulse. During the irrigations, however, the blood-vessels dilate and contract to a considerable degree at very short intervals. After the irrigations there is a rather rapid decline in blood-pressure, with a corresponding rise in pulse-rate. (See protocols 18 to 25, at the end of this chapter.)

Very hot and very cold irrigations, like very hot or very cold baths, have for their immediate effect an elevation of the blood-pressure. Irrigations at temperatures within the neutral limits, i.e., a few degrees above or below 98.4° F., have little effect on the circulation unless given under high pressure, when the blood-pressure will be raised just as in hot or cold irrigations. (See protocol 23b.) Similarly, playing a strong light on the eyes (protocols 18a and 18b), or any powerful stimulation of sensory nerves, raises the blood-pressure. During the irrigations the blood-pressure fluctuated considerably, and so rapidly that the figures in the protocols do not represent the changes fairly. One such instance of a steep drop in blood-pressure is shown in protocol 22a, where there was a drop from 140 mm of Hg. to 80 during an irrigation at 61° F. Such fluctuations occurred so suddenly that usually there was not sufficient time to catch and record the pressures.

The changes in the circulation, therefore, resemble those that follow rotation, and are produced by irritation and exhaustion of the same mechanisms. The vaso-motor system is in a constant state of active variation, readily recognized in the radial pulse and in the vessels of the fundus oculi.

In bilateral irrigations the blood-pressure and pulse-rate are affected about the same as in single irrigations of the same temperature. (See protocol 23a.)

The effects of consecutive irrigations, both unilateral and bilateral, are shown in protocol 23. Both kinds of irrigations have about the same effect in raising the blood-pressure, whilst the pulse-rate remains stationary. The effect of a neutral irrigation in raising the blood-pressure when the fluid enters the auditory canal under pressure, is shown in protocol 23b. With the irrigating receptacle just sufficiently above the ear, to insure a flow, the effect on the pulse-rate and blood-pressure was nil, whereas, with the receptacle 4¾ feet above the ear, the blood-pressure rose to 130, the pulse-rate remaining stationary.

There was little noticeable effect on the respiration beyond the initial inspiratory gasp at the commencement of the irrigations. When exhaustion began to set in, or the blood-pressure fell very low, the respiratory rate was increased. (See protocol 20a.)

The Effect of Drugs in Aural Irrigations

The effect of atropin in aural irrigations was in every respect similar to its effect in rotations. The efficiency of the vaso-motor system was enhanced and the cardiac mechanism released from vagus control. Protocols 22b and 22c show the action of atropin and the response of the cardiac and vaso-motor mechanisms to cold irrigations. Atropin had the same effect in diminishing nausea and sickness referable to the stomach that it displayed in rotation sickness. It had no direct effect, however, in preventing or diminishing vertigo, nystagmus, or disturbances of equilibrium.

Adrenalin m iii intravenously had its well-known effect on the blood-pressure. Its effect in contracting the blood-vessels was well demonstrated in the small vessels which lie along the handle of the malleus. These were quite invisible after the adrenalin, just as they are immediately after hot and cold irrigations. When the adrenalin effects had worn off, the small vessels reappeared and were then seen to be somewhat dilated, as after aural irrigations. (See protocol 23c.)

Adrenalin was without effect in combating the vertigo, nausea, nystagmus, and disturbances of equilibrium that are caused by aural irrigations. A 3% solution of cocain, in equal parts of analin oil and alcohol, applied to the interior of the external auditory canal and to the drum membrane, was without effect upon the phenomena of irrigations. Pilocarpin was tried and found to have no effect in preventing nystagmus, vertigo, nausea, gastric disorders, or disturbances of equilibrium. Gr 1/6 of the hydrochlorid was given hypodermically. In seven minutes the forehead was moist, and in fourteen minutes it was thickly covered with beads of sweat. Irrigations were then given with the subject in the supine posture. The only beneficial effect noticed was that the noise from the irrigation seemed to be less than usual, but as this frequently occurred where no drug had been used it seems we are not justified in crediting even this to the action of the drug. After the administration of the pilocarpin there was a gradual rise in pulse-rate and blood-pressure, followed later by a gradual subsidence, so that in the course of two hours the circulation was about in the condition it had been in before the administration of the drug. (See protocol 24.)

It will be noted that the effect of pilocarpin upon the circulation in this instance differs from that generally ascribed to the drug. Usually pilocarpin slows the heart by stimulation of the cardiac vagus endings, and lowers the blood-pressure by depression of the vaso-constrictor centre in the medulla. The absence of the usual effects of the drug upon the circulation in this instance may be explained by, (1) the moderate dosage, although the effect upon the salivary and sweat glands was pronounced; (2) the counteracting effect of the psychic condition and of surrounding circumstances (the subject had been a long time anticipating this test and during the experiment he lay on the table with very little clothing on him, as the day was warm); (3) the idiosyncrasies of the individual who seldom sweats, and never profusely, and whose circulatory mechanism is strong at the cardiac end, and perhaps relatively feeble at the vaso-motor end.

The evidence brought forth by these experiments does not tend to support those clinicians (Politzer, Lermoyez, and others) who claim so much for pilocarpin in inflammatory and other conditions affecting the internal ear.

The effect of hypnotic suggestion upon the phenomena evoked by aural irrigations was studied extensively. Following are some of the notes taken during the experiments: Irrigation at 72° F. of the left ear for three minutes in a subject with normal hearing in both ears caused the usual phenomena with nystagmus and apparent movement of objects to the right. In waking hypnosis, with the suggestion that it would not make him sick, dizzy, etc., another irrigation at 72° F. was given. The head did not seem to fall one way or the other. The subject was made very dizzy, but stated he did not feel the "loss of the supports" of the head on either side. There was slight visible nystagmus, but the eyes were kept closed and turned strongly upward, whilst the subject manifested a strong tendency to sleep. After the irrigation, and whilst still in hypnosis, the subject walked without staggering, but the gait was slow and studied, every step being apparently made with the greatest possible care. The subject was rather quickly taken out of hypnosis and at once lost his balance.

A little later, with eyes closed, he tried to stand first on the left foot, and then on the right. In each case it was impossible for him to steady himself. He was compelled to make a constant succession of hops in order to maintain his equilibrium. When attempting to stand on the right foot the hops were made toward the right, and when attempting to stand on the left foot the hops were made toward the left. The same subject, on a subsequent occasion, received an irrigation in the left ear at 70° F. but without hypnosis. The usual phenomena were manifested. After the irrigation, on attempting to stand on either leg the subject fell toward the left, but after a time there was a slight reactionary tendency to fall to the right.

Under hypnosis, with the suggestion that the irrigation would not disturb him, an irrigation of 70° F. for four minutes was given in the left ear. The visible nystagmus was not nearly as pronounced as might have been expected from such an irrigation. After the irrigation the subject, still in hypnosis, balanced well on either foot.

A prolonged irrigation at 70° F. was given under hypnosis, with the suggestion that it was warm water. The subject manifested great comfort in his facial expression, but at times made the usual wry faces. Nystagmus to the right was manifested, but it was not so apparent as one should expect. After the irrigation, and still in hypnosis, the subject stood well with the feet close together, and with eyes closed, but he was unable to balance on one foot with eyes open or closed.

It was noted that during the irrigations the left pupil was smaller than the right. The knee-jerks were slightly increased. Under the suggestion that the knee-jerks were exaggerated, the motor response to the tendon tap was slow and of wide range, suggestive of voluntary extension of the leg rather than of the sharp response of the true reflex.

On another occasion the same subject was put through balancing tests before irrigation was practised. It was found that with the eyes open he could stand well with both feet close together and on either foot, whereas, with the eyes closed he could stand on both feet, but not very well on either foot alone. The pupils were observed to be rather small. Before irrigation, the pulse-rates were 60, 72, 72, with corresponding blood-pressures of 120, 120, 115. (See protocol 25.)

Irrigation at 69° F. in left ear without hypnosis caused the usual phenomena. During the irrigation the pulse-rates were 68, 68, with blood-pressures of 120 and 120. After the irrigation, with the eyes open he could not stand well on either foot, but tended to fall to either side.

Under waking hypnosis the pulse-rates were 68, 68, with blood-pressure of 115 and 115. With the suggestion, that he was receiving a cold irrigation in the left ear, the subject made wry faces and behaved as if he were actually receiving a cold irrigation, although no irrigation whatever was given. Both pupils were moderately dilated, but the left was smaller than the right. There was no hippus present. On looking to the right, the eyes jerked slightly that way, and the head inclined toward the left. There seemed to be an awkward attempt at reproducing an irregular type of nystagmus.

Before irrigation the pulse-rate was 64, and the blood-pressure 115. An irrigation at 69° F. was given in the left ear for three minutes, with the subject in hypnosis, and with the suggestion that it was a pleasant, tepid irrigation which would not disturb him or make his eyes jerk. One minute after starting the irrigation the pulse-rate was 64, and the blood-pressure 115. There was scarcely any nystagmus present, and that chiefly on looking to the right. The head did not fall to the left after the irrigation, and the subject walked well, even with the eyes closed. The gait, however, was painfully slow and studied. On being taken out of hypnosis he stood and walked well, but he relapsed so quickly into hypnosis that it was doubtful whether he had been thoroughly aroused from the hypnotic state.

An irrigation at 68° F. was given in the left ear, but without hypnosis. The usual nystagmus, etc., were manifested. After the irrigation, the subject stood well with the eyes closed, and appeared to stand better on the foot of the side irrigated than on the opposite foot. In the last instance he fell generally toward the side of irrigation.

Under hypnosis a cork was placed in the subject's outstretched hand, with the suggestion that it was a heavy weight. Appropriate grunts and gestures were made as though a heavy weight was sustained at a great disadvantage, but little, if any, perceptible change in the pulse-rate or blood-pressure was registered.

On another occasion the subject was tested before irrigations were practised. With closed eyes he stood fairly well on either foot, but lost his balance after three seconds. Standing on the left foot, he lost his balance toward the left. Standing on the right foot, he lost his balance toward the right.

An irrigation of the left ear at 69° F. caused marked nystagmus in less than one minute. After the irrigation the subject, with eyes closed, was unable to stand on either foot. This disability persisted for one or two minutes.

Irrigation of left ear at 111° F. caused some nystagmus. Irrigation at 116° F. caused well-marked nystagmus. After the irrigation the subject was unable to stand on either foot with the eyes closed. After an irrigation in the left ear at 120° F. the subject, with eyes open, stood better on the right foot than on the left. With eyes closed, he was unable to stand on either foot, but made better attempts to stand on the right. The subject, after a short rest, was hypnotized and an irrigation at 69° F. of the left ear given, with the suggestion that the water was tepid and soothing, and would not make the eyes jump, etc., or disturb his balance. Violent nystagmus was manifested early during the irrigation. After the irrigation, with eyes closed, he balanced well standing on both feet, but did so very awkwardly on one foot, making wide sweeps of the opposite leg in order to maintain his balance, and being finally compelled to touch the floor.

Using the same subject, before irrigation the pulse-rates were 84, 92, 84, and 84, with corresponding blood-pressures of 110, 110, 110, and 110. An irrigation of the left ear was given at 69° F. During the irrigation the pupils were moderately dilated, the left being smaller than the right, whilst the pulse-rates were 76 and 76, the blood-pressures being 115 and 115. After the irrigation, the subject was dull and heavy. He had evidently gone into hypnosis. He was unable to stand with eyes closed, and could not lift one foot from the ground without falling.

Under hypnosis before irrigation, the pulse-rate was 76 and the blood-pressure 115. An irrigation at 69° F. was given in the left ear under hypnosis, and with the suggestion that the water was tepid and would not sicken or disturb him. During the irrigation the pupils at first contracted and then became moderately dilated, but the left remained smaller than the right. There was well-marked nystagmus to the right. During the irrigation the pulse was 76, and the blood-pressure 120, with the artery well contracted. After irrigation the subject balanced much better than he did after the previous irrigation, and yet both irrigations were of the same temperature and duration. However, it was only with great difficulty that he could stand on one foot.

Before being put into general cataleptic rigidity, the pulse-rate was 72 and the blood-pressure 110. During general cataleptic rigidity the pulse-rate was 76 and the blood-pressure 110. The suggestion was made to the subject that he supported upon his outstretched hand a weight of 100 pounds. During the efforts he made to support this imaginary weight the pulse-rate was 80 and the blood-pressure 115.

On another occasion, examination of the subject before the irrigations and without hypnosis showed the pulse-rate to be 84, 84, 88, and 88, the blood-pressures being 115, 115, 115, and 110. Under hypnosis the pulse-rates were 76, 76, 84, 88, and the blood-pressures 115, 115, 120, and 115. An irrigation of the left ear at 115° F. was given under hypnosis, and with the suggestion that the water was warm and agreeable and would not cause "jumping of the eyes" or other disturbance. During the irrigation the pupils were moderately dilated, but the left was a little smaller than the right. The head was inclined to fall to the right. There was no visible nystagmus. During the irrigation the pulse-rates were 76, 80, 80, 88, and the blood-pressures 110, 110, 110, and 105.

An irrigation of the left ear at 120° F. was given under hypnosis, and with the suggestion that it would sicken him and make him vomit. The subject made all sorts of attempts to vomit, but there was no actual vomiting. During the irrigation, with the attempts at vomiting the pulse-rates were 84, 88, 88, whilst the blood-pressures were 110, 130, and 135.

An irrigation at 64° F. in the left ear was given, with the suggestion that it would sicken the stomach and cause vomiting. There were occasional attempts at vomiting. During the irrigation the pulse-rates were 84, 76, 84, 84, 84, 96, 92, 84, 84, 84, 84, 92, and 92, with corresponding blood-pressures of 125, 115, 125, 125, 130, 115, 115, 115, 115, 115, 110, 105, and 105.

After an irrigation of left ear at 64° F. the subject was immediately put into general catalepsy in the standing position, with the feet a little apart, so as to insure proper balance. In this condition he was more easily pushed over to the left than to the right.

An irrigation of the left ear was given at 123° F. under hypnosis, but without any particular suggestion. There was no nystagmus, even on looking to the right or left. After the irrigation, on being made rigid he was stiff toward either side as he sat on the stool, but on being made rigid in the standing position he was easily pushed over to the right. He could not readily be pushed over to the left, but it took very little force to make him lose his balance toward the right. In the latter direction, on being pushed the subject toppled over like a statue. He seemed to be unable to make any effort at recovering his balance when his equilibrium was displaced to the right.

During irrigation of left ear at 62° F., under hypnosis, with the subject standing and in a state of general cataleptic rigidity, there was nystagmus toward the right, and the subject at first toppled over to the right, then to the left, and later on in various directions. His feet were close together.

An irrigation of the left ear at 62° F. was given the subject as he stood with the feet wide apart and the neck and head in a state of cataleptic rigidity. There was nystagmus to the right, but the head did not incline to either side. On closing the eyes, the subject felt himself revolving from right to left (probably an error) about his long axis. The irrigation was repeated with the subject rigid from the head down, but with the head and neck loose and free. During the irrigation the head fell freely toward the left side.

A bilateral irrigation at 62° F. was given, with the subject in general cataleptic rigidity and standing in a natural attitude. During the irrigation the subject at first fell backward and had to be supported. Later he was inclined to topple over in various directions. After the irrigation, standing with eyes closed and with the feet close together, he fell forward and to the left, but later he fell backward and to the right. It was found on investigation that the water used in the left ear was two or three degrees colder than that used in the right. It was quite some time after the irrigation before the subject could walk well. Some genuine attempts at vomiting were made after the irrigation, and later when the stomach tube was passed the subject actually vomited. This was an unusual thing, for previously it had been impossible to upset the stomach by irrigations. Evidently giving the irrigations with the subject standing and making the extra efforts necessary to preserve equilibrium, had something to do with sickening the stomach. The chyme vomited amounted to ℥ viii. It appeared to contain chiefly bread and curdled milk, the remains of a meal of bread, butter and milk, ingested at 7 a.m. The stomach contents were withdrawn at 10 a.m. Analysis showed: total acidity .32% by weight, free HCl .12%, combined HCl .12%. There was a considerable amount of mucus, but no bile or blood. Starch and protein digestion was fair. Lactic acid was present, due probably to the milk ingested. It should be noted that this was not a regular test meal.

From the foregoing, it appears that by means of hypnotic suggestion the disturbances of equilibrium following aural irrigations can, to some extent, be compensated. It is also evident, as seen from protocol 25, that in the main, hypnotic suggestion seemed to be able to counterbalance the usual effects of aural irrigations upon the blood-pressure. However, the natural resistance of the individual in this respect must be taken into consideration, since, on many occasions, irrigations in the normal waking state were without marked effect upon the circulation. It is also fairly evident that hypnotic suggestion cannot altogether prevent nystagmus during irrigations. The effect of posture upon the phenomena of nausea and vomiting was strikingly evident in this particular individual, who could not be made to vomit by irrigations or suggestions so long as he was allowed to remain sitting, but who was easily sickened by irrigations practised on him whilst standing erect. It seemed that where disturbances of equilibrium were compensated by hypnotic suggestion there was subsequently some tendency to physical exhaustion. In some instances it was noted that after repeated irrigations there was absence of response on the part of the labyrinthine receptors or their related mechanisms. This was taken as an indication of exhaustion. A similar condition was observed where aural irrigations and rotations were practised simultaneously or in immediate succession. It should be borne in mind that aural irrigations are not as effective in producing labyrinthine phenomena as rotations. In addition to this, the varying density, thickness, etc., of the portions of the temporal bone forming the roof of the external auditory canal and of the tympanum in various individuals makes the question of susceptibility to irrigations a particular one for each individual case. Hence, too much importance should not be attached to hypnotic suggestion as a means of counteracting irrigation sickness, since it had no marked effect in offsetting or preventing the distressing effects of rotations.

The conclusions are:

1. That in their general effects, aural irrigations act in a manner precisely similar to rotations, causing identical phenomena by disturbance of the self-same organs and mechanisms.

2. That bilateral simultaneous stimulation of the labyrinthine receptors of moderate intensity has less effect in causing the general characteristic nystagmus and deviations than unilateral stimulation of the same degree, although the effect of both upon the circulation and the mechanism of vomiting is about the same.

3. That atropin, adrenalin, pilocarpin and cocain (the last applied to the external auditory canal and drumhead), have no effect in preventing or suppressing the immediate effects of thermic irritation of the labyrinthine receptors upon the cerebellar and medullary centres, although some of the remote secondary effects (e.g., gastric disturbances) may be counterbalanced by atropin.

4. That the characteristic phenomena caused by aural irrigations are not due to circulatory disturbances in the labyrinth, such as increased blood-pressure or vaso-motor disturbances, because during the height of its action adrenalin did not prevent the usual effects of aural irrigations.

5. That hypnotic suggestion cannot overcome the primary effects of vigorous stimulation of the labyrinthine receptors, though it may help in preventing, for a time, displacements of equilibrium, and offset the effects of secondary disturbances, such as psychic distress and the like.

6. That the upright position (active equilibration) has much to do in determining certain of the effects, e.g., vomiting, etc., of aural irrigations, whilst the sitting posture diminishes or delays them.

PROTOCOLS

18a—On "S," January 10, 1909

Pulse-rate.	Blood-press.	Remarks.
68	95	Normal, sitting.
72	105	"
68	100	"
68	105	"
68	105	"
72	125	On stimulation of retina by strong light.
76	125	" " "
76	130	" " "
72	105	Light removed.
72	105	1 min. after.
76	95	2 "
68	100	3 "
72	100	4 "

On second application of the light the blood-pressure rose momentarily, but soon fell to 90.

18b—On "S," January 17, 1909

72	95	Normal.
68	100	"
68	95	Strong light thrown into eye
64	105	During stimulation of retina by strong light.
72	90	" " " "
72	100	" " " "
68	90	" " " "
68	80	After removal of light.
64	80	" "
64	80	" "
68	80	During stimulation of retinæ by light.
64	90	" " " "
64	90	" " " "
64	80	After removal of light.
64	80	" "

18c—On "S," January 10, 1909

68	115	1 min. after irrigation of left ear at 75° F. for 2 min.
68	105	2 min. after irrigation of left ear at 75° F. for 2 min.
68	95	3 min. after irrigation of left ear at 75° F. Dizzy.
72	100	4 min. after irrigation. Dizziness gone.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	64	120	Just after irrigation of right ear at 75° F. for 3 min. Dizzy.
	68	105	1 min. after irrigation.
	72	100	2 "
	68	100	3 "
	68	110	After irrig'n of right ear at 115° F. for 3 min.
	68	105	1 min. after. Slight dizziness.
	72	105	2 "
	68	...	After irrigation of right ear for 2 min.
	64	...	1 min. after. No dizziness until now.
	68	...	2 " Dizziness gone.
	68	...	3 "
	68	...	4 "

19—On "S," January 24, 1909

	72	115	Normal.
	72	115	"
	72	120	Strong light in eyes.
	72	125	" " "
	72	120	Light removed.
	72	120	1 min. later.
	72	110	Normal.
	72	110	"
	72	125	Just after cold irrigation of left ear.
	72	120	1 min. later.
	68	120	After cold irrigation of left ear.
	72	125	1 min. after.
	76	125	2 "
	68	115	3 "
	72	110	4 "
	72	110	5 "
	68	105	6 "
	68	105	7 "
	64	125	After cold irrigation of left ear.
	72	125	1 min. after.
	76	120	2 "
	68	115	3 "
	72	110	4 "
	68	115	5 "
	68	110	6 "
	64	115	After hot irrigation of left ear.
	72	115	1 min. after.
	72	115	2 "
	76	115	3 "
9:38	..	..	Atropin, gr 1/100, hypo.
9:39	72	105	
	72	110	
	72	100	
	68	105	

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
9:44	64	110	
	68	110	
	72	110	
9:48	72	110	
	72	100	
	64	105	
	68	95	
	64	95	
	64	105	
	68	105	
10:01	64	120	After cold irrigation of left ear.
	64	115	1 min. after.
	60	105	2 "
	64	110	3 "
	68	110	4 "
	64	110	5 "
10:10	64	125	During hot irrigation of left ear.
	68	110	1 min. after.
	68	110	2 "
	68	95	3 "
	68	105	4 "
	64	120	Stimulation of left retina by strong light.
	68	120	" " "
	72	115	Light removed.
	76	110	"
	76	115	Alternate stimulation of each retina.
	72	115	" " " "
	72	115	Light removed.
	..	105	1 min. later.

About one minute after the commencement of cold irrigations the subject became dizzy, and external objects seemed to move in the horizontal plane toward the subject's right. This phenomenon was found to be constant in all cold irrigations below a certain degree. The dizziness seemed to last until the blood-pressure began to fall. After atropin the pulse was small and irregular in rhythm and the cold irrigations seemed to have greater power to cause dizziness. The hot irrigations caused less dizziness than the cold, and the apparent movement of external objects was toward the subject's left, with an up and down element of motion. All irrigations were given in the left ear. The subject was nauseated once during the stimulation of the retina by strong light. After atropin the cold irrigations caused external objects to move (apparently) up and down with a wavy motion as well as to the subject's right.

20a—On "S," January 26, 1909

Pulse-rate.	Blood-press.	Remarks.
72	105	After smoking. Respirations 18.
76	120	" "
76	125	" "
76	125	" "
80	120	" "
80	120	" "
76	125	After irrigation of left ear with warm water. Respirations 18.
76	125	After irrigation of left ear with warm water. Respirations 18.
76	125	After irrigation of left ear with warm water. Respirations 18.
80	125	After irrigation of left ear with warm water. Respirations 18.
76	120	
80	115	
76	120	
80	120	
76	120	
76	120	After irrigation of left ear with warm water. Respirations 18.
76	120	After irrigation of left ear with warm water. Respirations 18.
76	120	After irrigation of left ear with warm water. Respirations 18.
76	125	After cold irrigation of left ear. Resp. 24.
80	125	" " "
80	125	" " "
80	115	" " "
76	120	" " "
72	115	" " "
76	110	" " "
76	120	" " "
76	120	" " "
80	130	After hot irrigation of left ear.
76	125	" " "
84	120	" " "
76	115	" " "

The warm irrigations had little effect upon the blood-pressure, pulse-rate, or respiration. There was no dizziness or nystagmus. The subject experienced nothing but a soothing sensation. The cold irrigations caused, at first, a rise in blood-pressure and in the pulse-rate. Later on, the blood-pressure and pulse-rate fell and showed a tendency to fluctuate. The cold irrigations, however, caused increased respiratory rate, dizziness, pallor, nausea, lump sensation in the stomach and nystagmus, with apparent movement of external objects to the subject's right. The hot irrigation caused dizziness and nystagmus, but external objects seemed to move toward the subject's left.

20b—On "S," January 29, 1909

Pulse-rate.	Blood-press.	Remarks.
76	115	Normal.
80	110	"
80	110	"
76	115	After warm irrigation of right ear.
80	110	" " "
80	115	" " "
..	125	During cold irrigation of right ear.
..	120	" " "
76	115	" " "
80	120	1 min. after.
80	120	2 "
80	120	3 "
76	120	4 "

The only effect of the warm irrigation was a soothing sensation. The cold irrigation sent the blood-pressure up, but there was a marked immediate drop during the irrigation not shown in the blood-pressure figures. In addition, there was dizziness, nausea, increased saliva and nystagmus, with apparent movement of external objects toward the subject's left. After the irrigation the subject had chilly sensations along the spine, and felt as if he "had swallowed smoke." With this there was a sense of oppression referred to the epigastrium. The hearing for the watch was 18 inches for both ears. The right external auditory canal was abnormally tortuous. The persistence of the blood-pressure at 125 after the cold irrigation was probably due to the nausea.

21—On "B," January 31, 1909

76	105	Normal.
80	105	"
76	105	"
76	105	"
76	105	"
76	115	"
76	115	"
76	115	After cold irrigation in left ear.
84	110	1 min. later.
68	125	After cold irrigation of left ear.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	84	120	1 min. after.
	76	115	2 "

After this irrigation there was a slight dizziness, especially when the subject raised or moved the head. During the irrigation the subject began to perspire, and a severe pain was felt in the left ear.

	72	125	After cold irrigation of left ear.
	72	125	1 min. after.
	76	115	2 "
	80	120	3 "
	76	110	4 "
	72	115	5 "

During the irrigation the subject experienced no discomfort or pain. After the irrigation no disturbance was felt so long as the subject kept his head still. On raising the head, or on looking up, the subject became dizzy and external objects seemed to move toward his right. On rocking the head from side to side the subject became sick and dizzy and experienced, when the head went toward the left, a strange sensation of dead weight on the left side, so that a distinct effort had to be made to drag the head back to the perpendicular. After the irrigation the subject felt weak and broke into a general sweat. He had the "lump-sensation" in the stomach and felt very depressed and wretched. The respirations were shallow and of normal or slightly increased rate. The subject evinced a marked inclination to lie down.

8:10			Atropin crystals, gr 1/60, hypo.
8:38	68	115	Normal.
	64	115	"
8:40	64	120	After prolonged cold irrigation of left ear.
	68	125	1 min. after.
	68	120	2 "
	68	120	3 "
	67	120	4 "
	68	115	5 "

During this irrigation the subject felt a pain extending from the left external auditory canal toward the larynx, and on the next day he developed a mild inflammation in the left tonsil. After this irrigation the dizziness was comparatively slight. There was no nausea, faintness, or sweating. There was no sense of dead weight in the left side of the head when the subject rocked his head from side to side. Vision was slightly disturbed, but the stomach felt well and the irrigation seemed to restore the subject's strength.

22a—On "S," March 7, 1909

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	68	115	After bilateral irrig. at 115° F. for 3 min.
	72	115	During irrigation of left ear at 115° F.
	68	105	After " "
	68	110	During bilateral irrigation at 100° F.
	68	105	During irrigation of left ear at 100° F.
	71	110	Normal.
	72	105	"
	72	105	"
	68	110	After ophthalmoscopic examination.
	60	110	During bilateral irrigation at 70° F.
	60	105	" " "
	72	105	After " "
	64	115	During irrigation of the left ear at 70° F.
	68	110	After " "

22b—On "S," March 28, 1909

	68	...	After irrigation of left ear at 61° F.
10:42			Atropin crystals, gr 1/45, hypo.
10:48	60	110	
10:49	52	105	
10:50	56	105	
10:59	64	100	
11:00	60	110	
11:06	72	125	
11:12	80	120	
11:13	80	125	
11:14	76	120	
11:15	84	130	During irrig. of left ear at 61° F. for 2 min.
	84	100	" " "
	84	105	" " "
	84	110	" " "
	80	115	" " "
11:32	80	120	Normal.
11:33	..	135	During irrig. of left ear at 61° F. for 2 min.
	..	140	" " "
	..	80	" " "
	..	125	" " "
	76	115	2 min. after irrig. of left ear at 61° F. for 2 min.
	..	105	3 min. after irrigation of left ear.

22c—On "S," April 2, 1909

10:42	..	..	Atropin crystals, gr 1/45, hypo.
9:30	60	115	Normal.
10:41	68	125	Hasty news of wife's illness.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
10:43	72	165	During irrigation of left ear at 60° F.
	..	120	After “ “ “
	56	115	Before bilateral irrigation at 55° F.
	68	125	30 sec. after “ “
	68	125	60 “ “ “
	68	120	After bilateral irrigation at 115° F.
	60	125	After irrigation of left ear at 115° F.

23a—On “S,” April 9, 1909

64	110	Normal, sitting.
64	110	“
60	115	Lying supine.
60	115	“
64	130	During irrigation of left ear at 65° F.
64	135	“ “
64	125	After “ “
64	115	“ “ “
64	135	During bilateral irrigation at 65° F.
60	115	Before irrigation of left ear at 110° F.
64	125	During “ “ “
60	110	Before bilateral irrigation at 110° F.
60	120	During “ “ “
60	120	During irrigation of left ear at 115° F.
60	110	Before irrigation of left ear at 65° F.
60	110	During “ “
68	120	“ “ “ Before vomiting.
..	135	“ “ “ After vomiting.

23b—On “S,” April 16, 1909

68	100	Normal before irrigation.
68	100	“
68	105	“
68	110	“
68	110	During irrig. of left ear at 101° F. under pressure of a few inches.
72	110	During irrig. of left ear at 101° F. under pressure of a few inches.
68	110	During irrig. of left ear at 101° F. under pressure of a few inches.
72	110	During irrig. of left ear at 101° F. under pressure of a few inches.
72	110	During irrig. of left ear at 101° F. under pressure of a few inches.
72	115	During irrig. of left ear at 101° F. under pressure of a few inches.
68	110	After irrig. of left ear at 101° F. under pressure of a few inches.

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Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	72	100	After irrig. of left ear at 101° F. under pressure of a few inches.
	68	100	After irrig. of left ear at 101° F. under pressure of a few inches.
	76	100	Normal before irrigation.
	72	100	“ “ “
	72	100	“ “ “
	68	120	During irrig. of left ear at 101° F. under pressure of 4¾ feet.
	68	130	During irrig. of left ear at 101° F. under pressure of 4¾ feet.
	72	130	During irrig. of left ear at 101° F. under pressure of 4¾ feet.
	64	105	After irrig. of left ear at 101° F. under pressure of 4¾ feet.
	64	100	After irrig. of left ear at 101° F. under pressure of 4¾ feet.
	60	100	After irrig. of left ear at 101° F. under pressure of 4¾ feet.
	60	100	After irrig. of left ear at 101° F. under pressure of 4¾ feet.
	64	100	After irrig. of left ear at 101° F. under pressure of 4¾ feet.

23c—On “S,” April 23, 1909

10:02	64	105	Normal.
	64	105	“
	64	105	“
10:04			Adrenalin chloride minims ii intravenously.
10:04½	56	130	Injection faulty. Much of drug lost.
10:05	56	105	
10:06	60	105	
10:09	60	105	Normal.
10:10			Adrenalin chloride minims iii intravenously.
	..	170	After adrenalin.
	..	170	“
	..	160	“
10:18	64	100	“

24—On “S,” June 20, 1909

8:42	68	100	Normal, lying supine.
	64	105	“ “
	64	105	“ “
	64	105	“ “
	64	105	“ “

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	68	100	Normal, lying supine.
	68	100	" "
4:00			Pilocarpin hydrochloride gr 1/6, hypo.
4:02	76	95	Saliva profuse. Fulness in head.
4:03	76	100	Radial artery large.
	76	100	
	80	105	
	80	105	
	80	100	
4:07	80	110	Radial artery smaller. Forehead moist.
	84	110	Pains in glans penis, as if he wants to urinate.
	80	110	
	80	115	
	80	105	
	84	105	
	84	110	
4:15	80	110	Forehead covered with sweat.
	80	120	
4:19	76	125	Cooled sweat ran into ear canal.
	76	120	
	84	115	
	84	120	
	84	120	
4:24	76	115	Sweat and saliva profuse.
4:25			Irrigation of left ear at 68° F. The irrigation caused nystagmus and the usual symptoms.
4:25	76	125	During irrigation of left ear at 68° F.
	76	125	" " "
	76	120	" " "
4:29	76	120	After irrigation of left ear at 68° F.
	80	115	" " "
	76	115	" " "
	72	115	Feels sick. On the verge of vomiting.
	72	100	
	76	105	
	72	105	
4:39	76	105	Pain has left penis.
	76	105	
	72	105	
	72	105	
	68	105	
4:45	72	110	During irrig. of left ear at 115° F. The usual phenomena appeared.
	76	115	During irrig. of left ear at 115° F. The usual phenomena appeared.
	72	115	During irrig. of left ear at 115° F. The usual phenomena appeared.
	68	105	After irrig. of left ear at 115° F. The usual phenomena appeared.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	68	105	After irrig. of left ear at 115° F. The usual phenomena appeared.
4:53	72	95	Feels sick. Distressed in stomach.
	72	100	" "
	72	105	" "
	68	110	" "
	72	115	" "
	68	110	" "
	68	110	Forehead dry.
	64	115	"
	68	105	"
5:06	68	110	During irrig. of left ear at 115° F. Usual phenomena.
	64	115	
	68	110	
	64	105	After irrig. of left ear at 115° F. Usual phenomena.
	64	95	After irrig. of left ear at 115° F. Usual phenomena.
	60	100	After irrig. of left ear at 115° F. Usual phenomena.
	64	105	After irrig. of left ear at 115° F. Usual phenomena.
	68	110	After irrig. of left ear at 115° F. Usual phenomena.
5:15	68	105	Resting.
	64	105	"
	64	110	"
	64	105	"
	60	110	"
5:23	60	105	"
	64	105	Pain referred to left of epigastrium.

Pilocarpin did not prevent the occurrence of the usual phenomena that attend aural irrigations, viz.: vertigo, nystagmus, nausea, etc. After the administration of the drug the pulse-rate and blood-pressure showed a tendency to rise, followed by a gradual subsidence to the normal. The irrigations affected the pulse-rate and blood-pressure much as they do where no drug has been previously administered, i.e., the blood-pressure was raised at first and later on lowered, the pulse-rate at first being lowered somewhat and later slightly raised.

25—On "F," Sept. 22, 1909

60	120	Normal.
72	120	"
72	115	"

Pulse-rate.	Blood-press.	Remarks.
68	120	During irrig. of left ear at 68° F. without hypnosis.
68	120	During irrig. of left ear at 68° F. without hypnosis.
68	115	Resting under waking hypnosis.
		An irrigation in the left ear at 69° F., under hypnosis and with the suggestion that the water was tepid, caused no distress and did not alter the blood-pressure.
68	115	During irrig. at 69° F. with suggestion that it was tepid water.
64	115	During irrig. at 69° F., with suggestion that it was tepid water.
64	115	During irrig. a 69° F., with suggestion that it was tepid water.
84	115	Normal.
84	115	"
88	115	"
88	110	"
76	115	Resting under hypnosis.
76	115	"
84	120	"
88	115	"
		Under hypnosis an irrigation at 115° F. was given in the left ear, with the suggestion that it was tepid water.
76	110	During irrig. at 115° F., with suggestion water was tepid.
80	100	During irrig. at 115° F. with suggestion water was tepid.
80	110	During irrig. at 115° F., with suggestion water was tepid.
88	105	During irrig. at 115° F., with suggestion water was tepid.

An irrigation was given in the left ear at 120° F. under hypnosis, with the suggestion that the water was tepid. The irrigation caused a rise in blood-pressure and pulse-rate, but the subject manifested no signs of nausea or sickness until it was suggested to him that the irrigation was making him sick, when he began to make forced attempts at vomiting, but no actual vomiting occurred.

84	110	During irrig. at 120° F., with suggestion that the water was tepid.
92	130	During irrig. at 120° F., with suggestion that the water was tepid.
92	135	During irrig. at 120° F., with suggestion that the water was tepid.

Pulse-rate.	Blood-press.	Remarks.
84	125	During irrig. at 120° F., with suggestion that the water was tepid.

An irrigation was given in the left ear at 64° F. under hypnosis, and with the suggestion that the irrigation would cause sickness and vomiting. Many attempts were made, but no actual vomiting occurred. In later tests vomiting was brought on by irrigations practised with the subject standing.

Pulse-rate.	Blood-press.	Remarks.
76	115	During irrig. at 64° F., with suggestion that it would sicken.
84	125	During irrig. at 46° F. with suggestion that it would sicken.
84	125	During irrig. at 64° F., with suggestion that it would sicken.
96	130	During irrig. at 64° F., with suggestion that it would sicken.
88	115	After irrig. at 64° F., with suggestion that it would sicken.
84	115	After irrig. at 64° F., with suggestion that it would sicken.
84	115	After irrig. at 64° F., with suggestion that it would sicken.
84	115	After irrig. at 64° F., with suggestion that it would sicken.
84	115	After irrig. at 64° F., with suggestion that it would sicken.
92	110	After irrig. at 64° F., with suggestion that it would sicken.
92	105	After irrig. at 64° F., with suggestion that it would sicken.

CHAPTER XVI

THE EFFECTS OF AURAL IRRIGATIONS UPON THE DIGESTIVE APPARATUS

Aural irrigations affect the stomach functions precisely as do rotations. Depending on the degree of irritation of the labyrinthine receptors, which in turn depends upon the variation from the neutral limits of the temperature of the fluid used, vertigo, nausea, increased salivation, abnormal sensations referred to the stomach, stasis of food content, failure of absorption, and vomiting are readily caused in rapid succession. Marked congestion also occurs, as evidenced by the vomiting of blood on a few occasions. It was a noticeable feature of aural irrigations and rotations that vomiting occurred with little warning and was of the projectile type. This made it necessary to interrupt the irrigations from time to time so that the test meal should be retained the proper length of time. In the following tests, after irrigating the stomach, the regular Ewald meal was given and allowed to remain in the stomach for one hour during which irrigations were given as frequently as the subject could tolerate them without vomiting. The amount of chyme removed after cold irrigations with the subject sitting upright averaged, in three test meals, ℥xiii. With the subject lying down, the amount of chyme removed was ℥viii in one test meal. In a control test meal in which no irrigations were given, the amount of chyme removed after one hour was ℥iv.

In cold irrigations, with the subject sitting upright, the K I absorption test showed the presence of iodin in the saliva in 32 minutes upon the average. In bilateral irrigations (one test meal), with the subject sitting upright, there was no reaction at any time for iodin in the saliva although the latter was much increased. In cold irrigations, with the subject lying supine, the saliva failed to show the presence of iodin all through the irrigations. In these irrigations the saliva was not increased. In a control test meal, the saliva reacted for iodin in 19 minutes. The apparent difference between the results of the absorption tests in aural irrigations with the subject sitting erect and in rotations may be partly explained perhaps, by the fact that frequent intervals of rest had to be allowed between irrigations whereas in rotations about the long axis the movements were constant though mild. However, the fact seems also to emphasize the greater efficiency of rotation in perverting gastric function. Where irrigations were prolonged, as in irrigations in recumbency, or where the degree of irritation was intense, as in bilateral irrigations, the tests failed to show any evidence of absorption.

The salol test for stomach motility showed that at no time, before or after the irrigations, was there any evidence that stomach contents had escaped into the intestine. However, a control test meal showed the presence of salol in the intestine in 2 hours and 32 minutes. In this test however, the salol had been given just after the meal. Given in this way it is quite possible that most of the food might reach the intestines before the salol. Grützner[104] has shown by feeding rats at different times with food of different colours, that the food first ingested lay next to the stomach walls, whilst the succeeding portions were arranged in regular concentric fashion. In this way most of the food first taken might reach the intestine, whilst part of it might find its way to the fundus and reach the intestine with, or perhaps after, food ingested at a later time.

From this it is evident that for purposes of testing gastric motility salol should be administered at the commencement, and not at the end, of the test meal. In the tests for stasis of gastric food content, failure of intestinal absorption might also be a possible source of error. In the experiments where the salol test showed no reaction for the presence of salicyluric acid in the urine, definite direct proof of excluding the failure of intestinal absorption is wanting. It was deduced, however, from general considerations, and especially from the fact that whenever there was evidence of propulsion of food into the intestine there was also evidence of active absorption, whereas on frequent occasions absorption was fairly active and yet everything pointed to complete stasis of the gastric food content.

In cold unilateral irrigations, with the subject sitting upright, the average in two test meals showed: total acidity .18% by weight; free HCl .11%, and combined HCl .05%. In one test meal the figures were: total acidity .18, free HCl absent, combined HCl .09. This was the occasion on which the subject vomited gross blood, evidence of which was also found in the chyme taken through the tube.

In cold bilateral irrigations, with the subject sitting upright, the total acidity was .14, the free HCl .09, and the combined HCl .05 (one test meal). In this instance the K I test failed to show iodin in the saliva which was abundant.

In cold irrigations, with the subject lying supine, the total acidity was .14, the free HCl .09, and the combined HCl .08. The saliva was not increased and did not react for iodin.

In a control test meal the figures were: total acidity, .26, free HCl .16, combined HCl .08.

In three meals consisting of bread, butter and tea, with milk and sugar, and allowed to remain in the stomach upon an average of one hour and 28 minutes the average amount of chyme removed was ℥iii. The average of the analyses showed: total acidity .26, free HCl .19, combined HCl .05.

In one meal, consisting of beefsteak, potatoes, bread, butter, cocoa with milk and sugar, and allowed to remain in the stomach 8½ hours, the amount of chyme removed was ℥iiiss. The figures were: total acidity .52, free HCl .28, combined HCl .16.

From the foregoing it is manifest that the pylorus is tightly closed in aural irrigations, except perhaps where they were practised with the subject in the supine posture when the salol test was not tried. General experience however, seems to show that even in such irrigations when they are prolonged, or of such a temperature as to beget a high degree of thermic irritation, the pylorus permits nothing to escape into the intestine. Since the foregoing was written this matter has been fully tried out. It was found that nothing escaped into the intestine when the irrigations were kept up fairly constantly and that absorption was retarded and the saliva diminished or not increased.

It is also manifest that in all aural irrigations the stomach acidity is relatively diminished. Where the amount of chyme and the saliva were increased, and the salol test negative, it is a question whether the diminished acidity was not due in part to increased salivary content. In a special test, the subject was instructed not to swallow any saliva during the irrigations. The result nevertheless was the same as regards lowered acidity, although there was a diminution in the amount of chyme.

With irrigations in the supine posture, the acidity was absolutely diminished. This points to a diminished production of acid, since in this instance, as in rotations with the subject lying supine, the saliva was not increased. At all events whatever the cause, for all practical purposes of digestion the acidity was reduced.

In irrigations with the subject sitting upright, absorption from the stomach was slightly delayed, whilst in bilateral irrigations with the subject sitting upright the saliva, though increased, did not at all react for iodin. This shows that increased stimulation completely inhibits absorption and perhaps stimulates the pylorus to a more tonic state of contraction.

The diminished salivation observed in irrigations in the supine posture is explained by the diminished activity of the otolithic apparatus in decubitus whereby fewer tonus impulses are sent from the labyrinth to the nausea and vomiting mechanisms and the closely related centre for salivary secretion in the medulla. The negative reaction of the K I absorption test may perhaps be attributed to the failure of salivary secretion, but this does not at all seem probable. In a later test, absorption was distinctly retarded.

The conclusions are:

1. That aural irrigations affect the digestive apparatus as do rotations and act upon the self-same mechanisms.

2. That, as in rotations, dorsal decubitus tends to prevent nausea, increased salivation and vomiting with the attendant psychic distress, but does not prevent the imme-

diate effects of labyrinthine irritation or depression upon the medullary and cerebellar centres whereby vertigo, nystagmus, closure of the pylorus, inhibition of normal gastric secretion, etc., are produced.

The Effects of Aural Irrigations Upon Equilibration

With the subject sitting upright a cold irrigation in the left ear at 70° F. if continued for a time varying from 2 to 4 minutes, will cause lateral inclination of the head toward the left shoulder and horizontal nystagmus directed toward the right with a subsidiary rotary element against the watch. If the subject keep the eyes closed during the irrigation, he will experience a sense of rotation about the long axis of his body from left to right. When the nystagmus is fully developed, external objects appear to move steadily from left to right with a slight tendency downward. After the irrigation, if the subject attempt to walk he will deviate toward the left, though after a time it is common to have a period when he may deviate toward the right. This is a reactionary stage corresponding to the after-nystagmus that occurs in rotations. With the inclination of the head toward the left, is experienced a sense of lightness or emptiness in the right side of the head and scalp, and a corresponding sense of dead weight and heaviness in the left side. After the irrigation, as the vertigo and nystagmus subside they can be revived by tilting the head from side to side, or by turning the eyes strongly upward or to the side to which the nystagmus was directed.

The effects of cold irrigations of the left ear can be readily recognized as similar in every respect to the primary effects of rotation about the long axis of the body from left to right.

In an irrigation at 115° or 120° F. given in the left ear, similar phenomena will be observed, only in this instance the lateral inclination of the head will be toward the right and the horizontal nystagmus directed toward the left with a rotary element with the watch, whilst external objects will appear to move from right to left and with a slight tendency downward.

The effects of hot irrigations of the left ear are thus seen to be identical with the primary effects of rotations about the long axis from right to left.

If a bilateral irrigation at 71° F. be given for from 2 to 4 minutes to a normal person sitting erect, and if care be taken to see that the temperature of the water used is the same on both sides, and that the fluid enters the auditory canal under equal pressure on either side, there will be no horizontal nystagmus or marked deviation of the head or eyes to one side more than the other, though the head on tilting may feel rather heavy on either side. There will, however, be a steady pull of the head forward on the chest, and external objects will appear to move up and down or away from the subject in the mesial plane. With these will be found a mild degree of vertical nystagmus of short range and directed toward the top of the head. During a bilateral irrigation at 71° F. with the subject in the supine posture and with the eyes open, the subject felt as if the couch were falling away from his body, i.e., as if he were rotating in the mesial plane "occiput first." If the eyes be closed when the nystagmus is in full activity, the subject will feel his body moving up and down in the mesial plane "see-saw fashion." During the nystagmus, turning the opened eyes strongly to one side or the other may develop a horizontal nystagmus with a rotary element. Turning the eyes up toward the vertex also seems to aggravate the vertical nystagmus toward the top of the head. The study of nystagmus in the irrigations under consideration is a matter of some difficulty and it is only after repeated trials in various postures that one can convince himself of the true direction of the ocular movements.

The general effects of cold bilateral irrigations resemble the primary effects of rotations in the mesial plane "occiput first" or the secondary effects of rotations in the mesial plane "face first." It will be remembered that in rotations in the mesial plane "face first" the after nystagmus was of short range and duration, and its direction difficult to ascertain.

Bilateral irrigations at 115 to 120° F. cause vertical nystagmus directed toward the feet. Objects seem to be moving from the head toward the feet in the mesial plane. The effects of these irrigations resemble the primary effects of rotations in the mesial plane "face first" or the secondary effects of rotations in the mesial plane "occiput first."

With the subject on the back and the head lowered so as to make an angle of 85° between the long axis of the body and the horizon, irrigations of the left ear gave the usual phenomena that accompany similar irrigations in the supine posture with the body in the horizontal plane.

Irrigations of the left ear at 71° F. with the subject lying on the left side caused the usual horizontal nystagmus toward the right with objects appearing to move in the same direction. If during the irrigation the closed eyes were directed, as though looking at an object straight in front of the face, the subject felt his body moving back and forth in the mesial plane, but the head at all times seemed to be lower than the feet. With the closed eyes directed to the left, the subject felt his body revolving in the mesial plane face forward. With the closed eyes directed to the right he felt his body revolving about its long axis from left to right. These phenomena show that turning the eyes strongly to one side or the other not only inhibits or enhances the movements of the characteristic nystagmus, but in the case of inhibition of the typical form tends to develop a latent vertical nystagmus. Thus turning the eyes to the right evidently exaggerated the horizontal nystagmus to the right which it is well known it always does, and the subject felt his body rotating about its long axis from left to right. Voluntarily fixing the eyes in front, tends also to inhibit the horizontal element directed to the right and partially develops the latent vertical nystagmus with its associated sense of motion of the head and feet backward and forward in the mesial plane. Turning the eyes strongly to the left completely inhibits the lateral horizontal element directed to the right, whilst it allows the latent vertical element free play which in this instance was directed toward the feet evoking the associated sense of motion of the body "face first" in the mesial plane. The causation of these phenomena will be discussed in a later chapter.

In irrigations of the left ear at 117° F with the subject lying on the left side, objects seemed to move from the feet toward the head with a slight deviation toward the right. With eyes closed and directed straight ahead, the subject felt his body revolving about its long axis from right to left. With the closed eyes directed to the left, he felt himself revolving about the long axis of his body from right to left. With the closed eyes directed to the right, he felt his body revolving in the mesial plane "occiput first." After the irrigation external objects seemed to move about his body and in front of his face over his head in the direction of the hands of the watch, i.e., in the coronal plane. Here again is evidence of the simultaneous presence of three forms of nystagmus, viz., vertical, horizontal, and rotary, any one of which may be enhanced at the expense of the other two by turning the eyes in the direction calculated to favor it. The fact that at the onset of the irrigation, external objects appeared to move from the feet toward the head is to be attributed to voluntary fixation inhibiting the horizontal, but not affecting the rotary nystagmus. Risien-Russell[60] found, after exclusion of the lateral movements of the eyes, that the only other movements represented in the frontal ocular areas of the dog and cat were the upward movements. It seems therefore that these animals, and perhaps those higher in the scale, have power to inhibit voluntarily side to side movements of the eyes and vertical movements directed from above downward, whilst the power to inhibit voluntarily other ocular reflex movements is wanting. There is evidence that in man the power of voluntary control of upward vertical, and of rotary movements of the eyes is less than in the case of the other movements.

This is evidenced by the fact that in most forms of vertigo, straining the eyes upward tends to aggravate the distress, whilst few if any have the power of turning the eyes voluntarily upon the horizontal antero-posterior axis.

It is also in part due to the feebleness of voluntary control of the upward movements of the eyes that rotation in the mesial plane with the "occiput first" causes such furious primary nystagmus and distress, whereas rotations in the same plane with the "face first" cause but little nystagmus or distress.

Irrigation of the left ear at 72° F. for 6 minutes, with the subject lying supine, caused the usual mixed nystagmus, with the horizontal element directed to the right, and the rotary element against the watch. With the eyes turned to the left during the irrigation, external objects seemed to move from the floor to the ceiling, i.e., in the mesial plane from behind forward. On looking straight ahead, objects seemed to move from left to right in a plane at right angles to the long axis of the body. On looking to the right, objects seemed to move from head to foot with a wheel-like motion in the coronal plane, i.e., against the watch.

When the eyes were closed and allowed to assume any position, the subject felt his body rotating in the coronal plane against the watch with an added slight up and down motion of the head and feet in the mesial plane, and a feeling that the head was lower than the feet. Every inspiration seemed to influence this up and down motion of the body. With eyes closed and turned to the left, the subject felt his body rotating in the coronal plane with the watch. He felt also a slight sense of rotation about the long axis from right to left, and his head seemed to be lower than his feet. With eyes closed and directed straight ahead, he felt his body rotating in the coronal plane, with or against the watch, and moving up and down in the mesial plane in seesaw fashion every time he breathed. With closed eyes turned to the right, he felt his head on a level with his feet, and also felt himself turning in the coronal plane against the watch, with or without a slight sense of rotation about the long axis from left to right. After the irrigation it seemed to the subject as if only the right side of his body was in contact with the couch, which is the equivalent of a tendency of the body to rotate from left to right upon the long axis.

Here, again, is seen the influence of turning the eyes in certain directions upon the subjective interpretation of disturbances in equilibrium. Especially noteworthy is the fact that turning the open (and closed) eyes to the right permitted the rotary nystagmus against the watch to overshadow the horizontal element toward the right, thus causing external objects to appear to travel in a circle in the coronal plane when the eyes are open, and giving the subject a sense of rotation of his body in the same direction when the eyes are closed. The phenomena are readily explained upon principles laid down in another chapter based partly on the arrangement and insertion of the ocular muscles. On opening the eyes during irrigations at 70° and 71° F. in the left ear, the following conditions were observed: With the eyes directed straight ahead, there was horizontal nystagmus with a rotary element toward the right and against the watch respectively; with the eyes turned to the left the horizontal nystagmus was at a minimum, and the rotary jerks seemed to be with the watch, or at least the jerks against the watch were considerably slowed down; with the eyes turned to the right, there was horizontal nystagmus to the right, with marked rotary nystagmus against the watch. At times the latter overshadowed the former.

In irrigations of the left ear at 118° F. for 5 minutes, with the subject lying supine, the following observations were made: With eyes closed and directed straight ahead, the subject felt his body revolving in the coronal plane with or against the watch, together with a sense of seesaw movement up and down from side to side about the long axis. With eyes closed and turned strongly to the left, the subject felt his body rotating in the coronal plane with the watch, together with a sense of rotation about the long axis from right to left. With eyes closed and turned to the right, he felt himself rotating in the coronal plane against the watch, but without any sense of motion about the long axis.

Irrigations were practised to show that the temperature of the fluid is the important factor in causing nystagmus and disturbances of equilibrium and not the force under which the fluid entered the auditory canal. Thus irrigations at 65° F. with the irrigating receptacle just sufficiently above the ear to insure a flow, gave the characteristic nystagmus, inclination of the head, etc. An irrigation at 66° F. was then given with the receptacle at 18 inches above the auditory canal. The results were the same as in the preceding, but perhaps a little accentuated. Irrigations at 101° F. were given with the receptacle just above the level of the ear and at a height of 4¾ feet above it. In neither instance was there nystagmus, displacement of the head, vertigo, or disturbance of equilibrium. With the fluid under a pressure of 4¾ feet, the blood-pressure was raised noticeably, and the vessels along the handle of the malleus immediately after the irrigations were much contracted, just as in hot and cold irrigations. From these experiments it is evident that the temperature of the irrigating fluid is the chief active factor in causing labyrinthine reactions in aural irrigations, the immediate rise in blood-pressure when the fluid enters the canal under pressure being an effect common to powerful sensory stimuli applied anywhere at the periphery. It is also evident that changes in the circulation are merely concomitants and not the cause of the labyrinthine phenomena.

Cool irrigations were given in the left ear, to determine a point well below the neutral temperature, at which even a prolonged irrigation had no effect in causing nystagmus, etc. Then the right ear was experimented with, to determine a point above the neutral point, at which prolonged irrigation did not cause nystagmus, etc. Thus safe limits were established for the left and right ears at 89° F. and 107° F. respectively. Irrigation of each ear separately with water at 89° F. and at 107° F. for 8 minutes caused no nystagmus or disturbance of equilibrium. A bilateral irrigation was now given with water at 89° F. for the left, and at 107° F. for the right ear, all other conditions and circumstances being the same as in the preliminary unilateral irrigations. Soon after the commencement of the irrigation there was mixed nystagmus directed to the right and against the watch, with external objects appearing to move rapidly to the right and downward. The head was displaced laterally to the left and the subject felt dizzy, nauseated and exhausted. These irrigations were repeated a number of times with similar results.

From these experiments it is clear that the labyrinthine disturbances depend for their causation rather upon the relative differences in temperature of the fluid used on either side than upon any absolute degree of heat or cold. This shows conclusively that a constant labyrinthine tonus is in operation controlling the muscular movements of the head and eyes, and also perhaps of much more extensive parts of the body. And since bilateral hot and cold irrigations do not cause horizontal nystagmus or lateral displacement of the head, it is evident that in aural irrigations the cause of the nystagmus and of the disorders of equilibrium is a disturbance of the normal irritability of the peripheral nerve endings (receptors), whereby the evenly adjusted balanced mechanisms that mediate labyrinthine tonus are temporarily deranged and normal impulses evoked by acts of equilibration or locomotion in one labyrinth or the other have an abnormal effect, depending upon the condition of the labyrinthine receptors in which they originate, i.e., whether the irritability of the receptors of one side is relatively increased or diminished as compared with existing conditions in the receptors of opposite labyrinth.

The vertical nystagmus and the displacement of the head forward and backward that occur in bilateral cold or hot irrigations rather support than contravene this view since, as shall be shown later, they depend upon depression or exaltation of the receptors of one limb (viz., those of the ampullæ of both superior semicircular canals) of the balanced mechanism controlling movements in the mesial plane, thereby relatively enhancing or depressing the irritability of the receptors of the opposed limb, viz., those of the ampullæ of both posterior semicircular canals.

In other words, bilateral cold irrigations tend to depress the sensory terminals, i.e., to heighten the threshold value in the receptors of the ampullæ of the superior canals, thereby lowering relatively the threshold value in the receptors of their functional opponents, viz., the posterior canals. Similarly bilateral hot irrigations tend to lower the threshold value in the receptors of the superior canals, thereby relatively raising that of the receptors in the posterior canals.

Besides the disorders already mentioned as following aural irrigations, disturbances of co-ordination were also observed at times. For movements not particularly involved in acts of equilibration, such as touching the tip of the finger to the nose, the co-ordination was perfect, providing the subject had a good basis of support, such as sitting on a chair, so that the body as a whole could be easily steadied without any great effort and thus supply a reliable fulcrum for the arm movements. At times in walking, the subjects' "footing was not sure," and the co-ordination for gross hand movements was impaired. The subject on walking after irrigations, frequently complained of a "sleepy feeling" in the legs, and a weakness about the knees. Occasionally in walking he erred in calculating the distance of his heel from the floor. How much of these symptoms was due to the nystagmus and general weakness it would be difficult to state, but it is certain that in general the co-ordination of the hands and feet, even in walking, was slightly, if at all, impaired. The position of the feet and hands in space was well recognized, the chief trouble being to keep the head perpendicular and so to preserve rectilinear locomotion, for the deviations in walking seemed to be due in most, if not all cases, to the deviation or displacement of the head and upper part of the body.

The knee-jerks showed no constant marked deviations from the normal. Occasionally there was slightly increased activity upon the side toward which lateral deviation of the head occurred. Sensation was unaffected, except for the paræsthesiæ already mentioned, and an extreme hyperæsthesia that frequently developed in the external auditory canal, noted especially in hot irrigations. So great was this at times, that the slightest touch of the irrigating nozzle was excruciatingly painful. In some subjects in whom the membrana tympani was intact, cold irrigations also, after a few minutes, caused such intense pain referred to the middle ear, that the irrigations had to be suspended.

The paræsthesiæ of the scalp and side of the neck and body that occur with irrigations have been already alluded to. They are described by the subject as a feeling of numbness or emptiness in one side of the head and neck, usually upon the side opposite to that toward which the lateral deviation of the head occurs. This is frequently coupled with a sense of "drawing" of the muscles (voluntary compensation) upon that side and a sense of dead weight or heaviness upon the side toward which lateral deviation of the head takes place.

Paræsthesiæ, referred to the homolateral half of the head, also occur when the external auditory canal is plugged, so that free access of air and liquids to the tympanic membrane and the adjacent area of the external auditory canal is prevented. This was done in order to determine whether cold irrigations of the external auditory canal without coming in contact with the drum membrane, and a small portion of the canal immediately contiguous to it, would cause nystagmus or disturbance of equilibrium. Under these circumstances irrigations in the left ear at 74° F. for 4 minutes were without effect. The only result was a peculiar emptiness or lightness all over the left side of the head accompanied by autophony. The feeling is well known to those who have had inflammatory exudations in the tympanum. It seems to be due to the relatively increased bone conduction whereby, on account of the diminished air conduction, impressions, ordinarily not heard, reaching the peripheral auditory apparatus on that side by way of the cranial bones become relatively exaggerated by exclusion of the tympanum as a means of communication of sound vibrations in the usual way.

Similarly the ear canals were plugged to study the effects upon equilibrium when the subject dived under water. There was no noticeable disturbance observed, but when only one ear canal was plugged, a sense of emptiness or lightness upon that side was experienced. This was accompanied by autophony. This seems to indicate that the cochlear receptors also mediate some kind of tonus, and that these receptors on either side of the body stand to each other in the relation of balanced mechanisms.

It was noticed on several occasions that in persons well beyond middle life and in those suffering from general arterio-sclerosis as well as those suffering from "nerve deafness," irrigations are not so potent in producing the characteristic disturbances as similar irrigations practised on younger individuals. In a case of locomotor ataxia, in the beginning of the ataxic stage, the sensitiveness of the labyrinthine mechanisms was distinctly increased.

In a person subject to gastric crises of tabetic origin, but in whom the stage of ataxia had not yet set in, hot and cold irrigations had but little effect. All forms of deafness seemed to be temporarily improved, especially by hot irrigations graded carefully to reach the point of efficiency. Little permanent good perhaps is to be expected in professional and senile deafness. Here the cause is due primarily to disease of the cochlear nerve as shown by Haberman and Alexander.[106] Nevertheless, since circulatory changes and disease of the blood-vessels are seldom wanting, and especially since absence of collateral anastomoses is one of the chief factors causing the "elective vulnerability" of the cochlear nerve, it seems that anything which offers hope of even temporarily improving the circulation in the internal ear, without adding, as many drugs undoubtedly do, to the danger of nerve degeneration, should be welcomed.

During and after aural irrigation of a degree sufficient to disturb the equilibrium, it was observed that, to resisted movement, the subject was much stronger on the side toward which the head inclined. Thus with cold irrigations of the left ear, slight force on the right side of the head was sufficient to displace it toward the left, whilst it took much greater force applied on the left side to displace the head toward the right. In the latter instance, the subject's muscles acted strongly and together, whilst in the former there seemed to be a want of combined action as manifested by inefficiency and jerky irregularities in the muscular contractions. Cold irrigations seem therefore to displace the head laterally by an impairment of function of the muscles of the opposite side of the body, which tend to hold the head in the erect position. Hot irrigations seem, on the other hand, to act by enhancing the muscular function upon the contralateral side.

The conclusions are:

1. That the phenomena of aural irrigations are, in all essential respects, analogous to those caused by rotations. Thus: (a) Irrigation of the left ear with cold water is equivalent to the primary effects of rotation about the long axis from left to right. (b) Irrigation of the left ear with hot water is equivalent to the primary effects of rotation about the long axis from right to left. (c) Bilateral irrigation with cold water is equivalent to the primary effects of rotation in the mesial plane "occiput first" (relative stimulation of the ampullary receptors of the posterior canals). (d) Bilateral irrigation with hot water is equivalent to the primary effects of rotation in the mesial plane "face first" (absolute stimulation of the ampullary receptors of the superior canals).

2. That reactionary secondary phenomena may occur after aural irrigations as after rotations, but that with the former their appearance is slower and much less manifest than with the latter.

3. That in aural irrigations, as in rotations, there are

various forms of subsidiary or latent nystagmus present, simultaneously with the type or combination which is dominant and characteristic for certain irrigations, and for rotations in a certain plane and direction; that by turning the closed eyes in certain directions, the dominant combination may be inhibited, thereby allowing one of the other forms of nystagmus to impress the subject, and so beget the subjective sense of motion usually associated with that particular type of nystagmus.

3. That the temperature of the irrigating fluid is the important factor in causing the labyrinthine phenomena in aural irrigations.

4. That in the lateral deviations of the head, cold irrigations act by depressing the function of the muscles on the opposite side of the neck and head, whilst hot irrigations act by enhancing the muscular efficiency or tonus on the contralateral side.

5. That each labyrinth exerts reflexly a nicely adjusted tonus on the related muscles that control the equilibrium of the head and upper part of the body; that aural irrigations act by alteration of the normal irritability of the labyrinthine receptors on one side or the other, whereby the evenly adjusted mechanisms of labyrinthine tonus on either side of the body are deranged, and afferent impulses, evoked by ordinary acts of equilibration or locomotion have an abnormal value, enhanced or the opposite; that as a consequence, abnormal responses on the part of the related muscles result, depending upon the relative condition of irritability that obtains in the labyrinthine receptors as compared with the condition of irritability of the corresponding receptors in the opposite labyrinth.

6. That the disturbances of co-ordination that accompany aural irrigations affect mainly movements of the body calculated to steady the upper portion of the body upon a lower fixed portion, and that consequently there is no true inco-ordination of the feet or hands.

7. That sensory stimulation of the external auditory canal is not the cause of the characteristic phenomena of aural irrigations.

8. That hot and cold irrigations act by increasing and depressing respectively the irritability of the labyrinthine receptors; that these effects are brought about by direct

conduction through the osseous structure situated just above the attachment of the membrana tympani where lie, in close proximity to the surface, the horizontal semicircular canal with its ampulla and the ampulla of the superior semicircular canal, the macula of the utricle being in close proximity.

9. That aural irrigations are accompanied by paræsthesiæ of the neck and scalp which are fairly characteristic and constant in their relations to the labyrinth affected, and which indicate probably impaired cerebellar function.

10. That aural irrigations are a possible aid in the treatment of ''nerve'' and other forms of deafness where little is to be expected from the ordinary methods; and that they may possibly help in arresting the progress of degeneration by enhancing the local circulation.

CHAPTER XVII

THE EFFECTS OF AURAL IRRIGATIONS UPON THE EYES

The effect of aural irrigations upon the pupils was studied. The results, however, whilst usually striking were by no means uniform. The sequence of the phenomena in cold irrigations (62°–75° F.) most frequently appeared to be as follows: The first effect immediately following impact of the fluid against the interior of the external auditory canal was dilatation. This was soon followed by constriction, first upon the side of irrigation and later upon the opposite side. After a period of from five to fifteen seconds both pupils again became dilated. From this point on, there was usually a condition of alternating dilatation and constriction (hippus), the average size of the pupils being that of moderate constriction. The hippus was studied under a strong light from the ophthalmoscope and in moderate daylight. In cold irrigations there was a tendency of the pupil upon the side of irrigation to constriction as compared with its fellow, though in some instances it was the larger of the two. During the irrigations there was congestion of the conjunctiva of both eyes, but more marked upon the side of irrigation.

Immediately following cold irrigations, the pupils in many instances became constricted on closing the eyes, but one minute after the irrigation the pupils did not contract but rather dilated somewhat on closing the eyes. The constriction of the pupils on closing the eyes accords with a general tendency to sleep during the irrigations when the eyes were kept open only with an effort.

In hot irrigations (115°–119° F.) the pupillary phenomena were similar to those in cold irrigations, but there seemed to be a tendency to relative constriction in the pupil on the contralateral side.

In a bilateral irrigation at 79° F. in the left, and 107° F. in the right ear, the left pupil was larger than the right. Closing the eyes seemed at times to revive the waning symptoms of disturbances, whilst at other times it tended to relieve them.

The above conclusions were based upon numerous observations made in the general study of irrigations. Later, a series of irrigations was undertaken with a view to a more specialized attempt in the study of the pupillary reactions. Irrigations were practised with the subject lying supine, and in such a position, that a moderate uniform daylight illuminated both eyes. In some of the irrigations, by carefully plugging the inner third of the external auditory canal with non-absorbent cotton, care being taken to avoid contact with the membrana tympani, an attempt was made to differentiate the effects of thermic stimulation of the sensory nerves of the canal from those due to labyrinthine stimulation. The pupils were observed, and rough drawings made from them before, at the onset of, during, and after each irrigation. The hippus was also studied, and an attempt made to measure the duration and extent of the alternating variations. The pulse-rate and blood-pressure were also observed during some of the irrigations. In some instances during the irrigations the pupil reactions in accommodation were also studied.

As the mechanisms of pupillary dilatation and constriction are somewhat complex, and as there is by no means a general uniformity of opinion as to their modus operandi, the observations made in this last series of irrigations are set forth somewhat in detail.

Irrigation at 117° F., with the ear partially stuffed—Pupils before irrigation were moderately dilated the left being somewhat larger than the right. At onset of irrigation the pupils were somewhat contracted, with the left a little smaller than the right. Later on the pupils grew still smaller and remained small and of equal size. During the progress of the irrigation there was hippus of very short range, and occurring at intervals of from 1 to 3 seconds. The pupils were smaller if anything, and equal, and did not dilate much in fixation for distance. One minute after irrigation the pupils were dilated almost to the degree observed before irrigation and now they were equal. The subject felt numb and cold in the left side of the head.

Irrigation at 117° F. with no stuffing in the ear. Before irrigation the pupils were moderately large, the left being slightly the larger. At the onset of irrigation the pupils showed no change for half a minute. During the irrigation the pupils were somewhat contracted the left being still a little the larger. The hippus cycle extended over a period of 4 seconds. Four-fifths of the time was taken up with the phase of contraction of the pupil and the resting stage before the next dilatation, whilst one-fifth of the time was consumed in the actual process of dilatation. The dilatation, therefore, was more active than the constriction, and its extent was about one-fourth of the width of the iris. At this stage the pupils were still smaller and equal. During fixation for distance, the pupils dilated somewhat and the range of the hippus was diminished. After irrigation the pupils began to widen, and the hippus ceased. Three minutes after irrigation the pupils were equal and about as large as they were before irrigation.

Irrigation at 75° F. without stuffing in the ear. Before irrigation the pupils were moderately large and equal. At onset of irrigation the pupils showed no change. During irrigation the pupils were slightly constricted, the left more so than the right. Intermittent nystagmic movements; no hippus present. Pupils contracted in accommodation for near vision; the subject was drowsy; the eyelids were constantly closing.

Irrigation at 65° F. with ear plugged. Before irrigation the pupils were moderately large, the left being the larger. At this time there was hippus of wide range. In the stage of constriction, the pupils still further contracted in fixation for near vision (accommodation) and dilated in fixation for distance, but only to an extent corresponding with that to which they had previously contracted for near vision. In other words, in fixation for distance, the pupil could not relax the constrictor effect to any extent, whilst in fixation for near vision, the pupil could contract still further. Fixation for distance seems to make the subject drowsy. The pulse-rate was 72 and the blood-pressure 105. At the onset of the irrigation, the pupils showed no appreciable change in size, though the blood-pressure rose to 115 with a pulse-rate of 76. During the irrigation the pupils showed little, if any, change in size. Hippus of short range was present.

Irrigation at 65° F. without plugging the ear. Before irrigation, the pupils were moderately large, the left being slightly the larger. There was some hippus present. The blood-pressure was 115 and the pulse-rate 80. At the onset of the irrigation, the pupils were constricted somewhat, the left more so than the right. Hippus of short range was present. The blood-pressure was 125, and the pulse-rate 80. During irrigation the pupils became still more constricted, the left being still the smaller. Hippus of short range was present. There was conjunctival congestion more marked in the left eye.

Irrigation at 119° F. with the ear stuffed. Before irrigation, the pupils were moderately large, the left being the larger. Each pupil contracted to light, but the left showed at times a tendency not to contract. The blood-pressure was 105 and the pulse-rate 76. At the onset of irrigation the pupils showed no change. Both contracted for near vision. During irrigation the pupils became somewhat constricted, the left being still the larger. One minute after irrigation, the pupils were about as they were before the irrigation.

Irrigation at 119° F. without plugging the ear. Before irrigation, the pupils were small and equal. Hippus was present and was of a twofold variety. Excursions of long range occurred every 5 to 10 seconds, the dilatation being executed in three or four bounds with the constricting phase setting in immediately on the completion of dilatation. Between these larger excursions, i.e., in the resting stage at the end of constriction, excursions of short range occurred each dilatation giving place to a constriction of equal range. Noise made by clapping the hands near the ear, caused the pupil to dilate to a slight extent. Just before the onset of the irrigation, the pupils were moderately small and equal. At the onset of the irrigation, the eyelids blinked, and the pupils dilated somewhat, being equal size. A moment later, the pupils were unchanged, except that the left was larger than the right. Hippus was present, but not as frequent or active as before irrigation. During irrigation, the pupils became small and equal, in spite of the fact that the subject had a sharp pain in the ear all through the irrigation. One minute after irrigation, the pupils were about the same as before irrigation.

Irrigation at 62° F. without plugging the ear. Before irrigation, the pupils were moderately large and equal, but changed and became small and equal just before the onset of the irrigation. At the onset of the irrigation, the pupils were moderately constricted and equal. There was little, if any, hippus before the onset of nystagmus. Closing the eyes caused dilatation of the pupil; opening them caused a sharp constriction. Toward the end of the irrigation, the pupils were moderately small, with the left somewhat the larger. Just after irrigation, the pupils were unchanged, but shading the eyes at this time caused a dilatation that was brisker than during the irrigation, and the constriction to light seemed to be less sharp. The subject felt no pain during this irrigation. After the irrigation he felt heavy and yawned much. Finally he fell asleep.

From the foregoing it appears that the immediate effect of both hot and cold irrigations, with and without plugging of the ear, was either nil or resulted in constriction of the pupils. It should be noted that, as a rule, the condition of the pupils before irrigation was that of dilatation. Where the pupils were small, one hot irrigation without plugging caused a slight dilatation, but not quite up to the degree that usually prevailed before the irrigations. Partial plugging of the ear, so as to keep the irrigating fluid from entering the inner third of the auditory canal, seemed to have no particular effect upon the immediate results of the irrigations. It had some effect, however, upon the results of prolonged irrigation. Thus in hot irrigations, with the ear stuffed, the pupils did not contract so much as in hot irrigations without stuffing; and hippus was absent or of slight range in the former case, whilst in the latter, it was generally present and occasionally of wide range. In one cold irrigation, with the ear plugged, the pupils did not contract or become unequal, whilst in several cold irrigations without plugging, the pupils almost invariably contracted somewhat and became unequal, that on the side of irrigation being the smaller. In cold irrigations the hippus seemed to be about the same; that is, slight and of short range with and without plugging of the ear. The inequality of the pupils seems therefore to belong to the phenomena of labyrinthine disturbance, and judging from the effect of cold irrigations in causing the pupil on the side of irrigation to become constricted it seems probable that the inequality of the pupils, seen at times in hot irrigations, is due to a relative constriction of the contralateral pupil. The changes in the blood-pressure and pulse-rate that occurred during the irrigations did not seem to bear any definite relation to the changes in the pupil. In one instance hippus of long range was present before irrigation. Here, however, it should be noted that the subject had already had seven irrigations. The hippus, therefore, in this instance may have been the result of previous irrigations. Judging from the time of its appearance and from its occurrence with and without plugging of the ear canal, it seems that the ordinary alternating movements of the pupil can hardly be attributed to the effect of the irrigations upon the labyrinthine receptors. However, it seems probable that where the range of excursion was extensive, the hippus was due to more than stimulation of the receptors in the auditory canal and must probably be classed among the labyrinthine effects. This form, which perhaps alone deserves the name of hippus, occurred only when the ear canal was not plugged, and generally some time after the onset of an irrigation. In the accommodation tests it appeared that no matter how small the pupils were, they could be made smaller by fixation for near vision, whilst in fixation for distance the range of dilatation never seemed to exceed the limits of the primary state of the pupil, i.e., before fixation for near vision was tried. During a cold irrigation it was seen that after shading the eyes the pupils reacted quickly on readmission of light, whilst after the irrigation the constrictor reaction was less active.

We may therefore conclude that, in general, hot and cold irrigations tend to cause unequal pupillary constriction, hippus, conjunctival congestion, a tendency of the eyelids to close, and a distinct inclination to sleep.

The tendency of the pupils to contract, even at the onset of cold or hot irrigations, is remarkable, because it is well known and accepted that stimulation of any sensory nerve, as well as of numerous areas of the cerebral cortex, is attended with pupil dilatation, whilst but few areas of the brain and cerebellum are associated directly or indirectly with pupil constriction.

During these tests the illumination was by daylight of moderate dulness. The subject was cautioned against fixing his vision for near or distant objects. All the irrigations were given in the left ear with the subject lying upon the back.

By way of contrast the pupils were similarly studied in rotations about the long axis. In general it was found that immediately on abrupt cessation of rotation from left to right the pupils first became moderately dilated and soon after contracted. Hippus was present of large excursion, the cycle covering a period of 7 to 8 seconds. Later the pupils became still smaller and the right always being the smaller, although at times the left pupil also became very small. Three minutes after a rotation, the pupils became almost as large as before rotation, and there was still some hippus present.

Upon cessation of a rotation from right to left the pupils dilated for a short time and later contracted. At this point hippus appeared with cycles of a duration of from 8 to 7 seconds. On closing the eyes the pupils dilated. Six and a half minutes after rotation the pupils were somewhat constricted, as compared with the normal before rotation.

At times, during aural irrigations and following rotations, great difficulty was experienced in observing the fundus oculi, owing manifestly to disturbances in the mechanisms of refraction. These changes are interesting because changes in refraction have frequently been noted in connection with the study of the physiology of the cervical sympathetic. Thus Morat and Doyon[108] found diminution in size of the anterior crystalline lens images after section, and enlargement of the same images after stimulation of the cervical sympathetic. Hess and Heine,[109] however, demonstrated that stimulation of the sympathetic dilates the pupil without altering the condition of the ciliary muscle, and attributed the errors of refraction to changes in the peripheral portions of the cornea and lens by dilatation of the pupil. Rohmen and Dufour[110] concluded that the alteration in refraction on dilatation of the pupil was not necessarily due to actual change in the lens, and that the sympathetic cannot cause negative accommodation. Terrien and Comus[111] showed that stimulation of the cervical sympathetic, after section, caused an increase in refraction of the eye from 1 to 2.5 D. This change sets in before dilatation of the pupil and is of shorter duration than the latter. These authors give no satisfactory explanation of the phenomenon.

From these results it is evident that stimulation of the cervical sympathetic in some way changes the refraction of the eye. It is probable that the changes in refraction caused by irrigations and rotations are similarly caused, but they were observed when the pupil was under the influence of homatropin just after rotations and during aural irrigations.

In discussing hippus it must be remembered that ordinarily when the intensity of light entering the eye is altered, the pupil contracts and oscillates rapidly, alternately overstepping the mark in contraction and in dilatation, until finally it settles down in contraction which is slightly less than that first exhibited. In abnormal conditions this normal oscillation of the pupil may be exaggerated as follows: (1) Where the excursions are wide, readily seen, and independent of increased intensity of light falling upon the eye. This condition is known as hippus. It is due to rhythmic activity of the nerve centres and is ordinarily not a peripheral phenomenon. (2) Where there is lack of sustained contraction under the influence of light. Here the pupil contracts sluggishly when the intensity of the light is increased and whilst the light is left constant the pupil slowly dilates and often with sluggish oscillations. This is a pathological phenomenon caused by diminished conductivity in the afferent paths of the light reflex, i.e., usually in the optic nerve, e.g., retorbulbar neuritis (Parsons[42]). With the hippus occurring during aural irrigations were associated at times the ordinary rhythmic oscillations of the pupil. In the latter the contractions and dilatations were evenly balanced and of about equal range and duration. In the larger excursions, without alteration of the light conditions, the dilatation was sudden and abruptly executed, whilst the contraction began immediately following the sudden dilatation, but was carried out so slowly that it was difficult to say when it ended. After closely observing this form of hippus it is hard to avoid concluding that in irrigations, etc., it results from hypertonus of the pupillo-constrictor mechanism which, from time to time, is momentarily overcome by the activity of the pupillo-dilator mechanism. Anderson[193] found that hippus frequently occurred after section of the third nerve, and that it was excited by tactual stimuli or by a certain state of anæsthesia. On section of the cervical sympathetic the hippus ceased. These observations, however, are quite compatible with the theory of reciprocal innervation between the dilator and constrictor mechanisms.

Waymouth Reed,[112] because of the electric phenomena manifested during contraction of the sphincter iridis, concludes that such a relation exists. Anderson,[112, 193] however, opposes this view, because: (1) after section of the third nerve variations in illumination do not affect the pupil; (2) paradoxical pupil dilatation may arise and last a minute in bright sunlight and (3) in the inequality of the pupils following paralysis of one dilator mechanism the inequality is not diminished but increased by bright illumination of the eyes. Inasmuch as the modus operandi of paradoxical pupil dilatation is not sufficiently understood, conclusions based on evidence derived from the phenomenon must be regarded with suspicion. Then again the conditions obtaining in the pupil after section of the third nerve or of the cervical sympathetic are such as tend to mask reactions from alterations in illumination. On the other hand Jessop[209] never saw, in the iris, dilator fibres to compare in thickness and strength with those of the sphincter. It is difficult, therefore, to understand how such a feeble dilator mechanism can, for instance in stimulation of the sympathetic, overcome the stronger constrictor muscle without the intervention of some such process as reciprocal innervation. And as reciprocal innervation is of such widespread occurrence and plays such an important rôle in bodily adjustments it may be accepted, pending further researches, that it is utilized in the mechanism of pupillary phenomena.

In the hippus that occurs with aural irrigations and rotations the question arises: Is it the result of pathic stimuli? v. Bechterew[210] associates the light reflex with the pathic or sensory, and showed that the dilatation in the latter was due to inhibition of the former. This seems to make the sensory dilatation as it were part of a nociceptive reflex. Anderson[193] agrees with v. Bechterew's view but says that there is at the onset of the reflex a rapid short dilatation due to augmented dilator tone. Inasmuch as the dilatation in aural irrigations usually commenced only after the lapse of a period sufficient to permit the irrigations to affect the labyrinthine receptors, and inasmuch as the state of the pupils in irrigations was, in general, that of unequal contraction rather than of dilatation, it seems that the hippus is to be considered as an associated labyrinthine phenomenon caused probably by subjective sensations attending the disturbances of equilibrium. The positive element of the dilating phase of the hippus seems due to increased tonus in the dilator mechanism. Such increase of tonus always follows inhibition and lowers the threshold value at the commencement of the final common path for the paths inhibited (Sherrington[65]). Additional factors in augmenting the dilator tonus are re-enforcement or bahnung and summation of stimuli, whether originating in the cerebral cortex or at the periphery. The negative element in dilatation is due to inhibition of constrictor tonus, the threshold for afferent constrictor impulses being raised at the commencement of the final common path, just as that for afferent dilator impulses becomes lowered. In this manner alternate changes in threshold values at the commencement of the final common paths give rise to the rhythmic dilatations and contractions of the pupil (alternating reflexes). The slowness of the constricting phase is to be accounted for by fatigue of the constrictor mechanisms. Anderson, after eliminating the dilator paths, saw similar evidence of fatigue in eliciting the sensory reflex. Repeated stimuli were without effect unless an interval of some minutes was allowed.

The foregoing explanation of hippus implies the existence of final common paths which unite the dilator and constrictor effectors in associated action. The co-ordinating centre is situated at the commencement of these associated final common paths somewhere in the cerebellum or mid-brain. As the simple reflex has been amalgamated with other reflexes to make the type reflex, so the pupil reflex in certain cases has been compounded with other nociceptive reflexes to form complicated protective movements. We have already seen that the simple direct connection between the oculo-motor and vestibular nuclei is not sufficient to insure co-ordinated action between ocular movements and movements of equilibration. A higher co-ordinating centre is necessary. Similarly, in certain phases at least, the pupil reflex seems to require some higher co-ordinating centre, exclusive of that within the third nucleus. Ferrier's[8] observations give us grounds for locating the centres for reflex ocular movements in the cerebellum. Ferrier's experiments also seem to indicate that there are pupillary centres in the cerebellum associated with those for ocular movements. The exact location of the higher pupillary centres is a matter for future research. Meanwhile it is justifiable to assume their existence presumably in the cerebellum or mid-brain, judging from analogy and the results of certain experiments in relation to the spinal reflexes. The paths described by v. Bechterew as passing in the superior peduncle between the third nucleus and the cerebellum are probably related to this higher representation of pupillary movements. See superior peduncle in the anatomical synopsis.

The possibility of alternating vascular changes, coincident with similar changes in the general circulation, causing the hippus phenomena, may be dismissed since the range and suddenness of the iris movements were altogether beyond the capacity of mere vascular variations.

The constriction of the pupils so regularly seen in aural irrigations results, to some extent perhaps, rather from the prolonged constrictor phase of the hippus than from any constrictor effect attributable to vestibular irritation. The inequality of the pupils results from relative or absolute increase of cerebellar tonus in one side as compared with the other. Ferrier[8] has shown that stimulation of one side of the cerebellum causes homolateral pupillary constriction. In cold irrigations of the left ear the labyrinthine receptors are depressed. As these receptors are mainly related to the opposite half of the cerebellum there is depression of cerebellar tonus upon the right side with a relative increase of it on the left side. This causes the relative constriction in the left pupil found so constantly in cold irrigations in the left ear. Similarly in hot irrigations in the left ear the cerebellar tonus is increased upon the opposite side, causing relative constriction of the right pupil.

The first effect of hot and cold irrigations upon the retinal vessels was dilatation. This was immediately followed by marked contraction. When the irrigations were continued until nausea, sickness and general exhaustion resulted, the retinal vessels were in a state of extreme contraction, the dark shadows from the chorioidal vessels showing plainly through the pale retina. A short time after the irrigations the retinal vessels were regularly somewhat dilated. At times the subject saw coloured images, evidently shadows from the disc and retinal vessels. Irrigations at 100° F. caused the retinal vessels to dilate.

These observations correspond closely with those made by Rockwell and Beard [107] upon galvanization of the sympathetic in the neck. Roosa, who held the ophthalmoscope, saw at first hyperæmia of the retinal vessels followed soon by anæmia. However, much difference of opinion exists between observers as to the precise changes in the retinal vessels that follow galvanization of the sympathetic. Some saw only contraction, whilst others saw only dilatation. Perhaps the difference in the findings is due to a difference in the strength of the stimulus and a difference in the time of observation as, after a certain length of time (a few moments) following aural irrigations, the retinal vessels became dilated. The constriction and dilatation of the retinal vessels during rotations and irrigations seem to be but a part of the general circulatory changes in progress during rotations and irrigations, and seem to be caused by alternate excitation and inhibition of the vasoconstrictor centre by stimuli originating in the labyrinthine, cardiac and other receptors.

The visual fields did not seem to be affected in any particular manner by aural irrigations, but no adequate tests were made upon which to base reliable conclusions as to this point.

The apparent movements of objects during the after-nystagmus in rotations and during the nystagmus of aural irrigations needs a little further elucidation. It has already been explained that in horizontal nystagmus, say toward the right, vision is impossible during the excursions of the fundus to the left under the influence of the short jerky elements of the anterior part of the eyeball to the right. Vision, however, is possible during the slow return movements of the fundus toward the right. It is owing to this slow return movement that images of external objects fall upon successive horizontal areas of the retina which are nearer and nearer to the left of the fundus. This movement of images upon the retina toward the left is falsely interpreted (projected) as a movement of external objects toward the right. See diagrams. A similar explanation holds for vertical nystagmus.

In rotary nystagmus, vision is likewise impossible during the rapid excursions at the height of the nystagmus. During the slow return movement, however, vision is clear. Thus in rotary nystagmus against the watch, the slow movement of the fundus occurs with the watch. During such a movement objects directly in front of the eyes casting their images directly on the centre of the retina do not seem to move, whilst objects to the left of the subject appear to move upward from the feet to the head and those on his right seem to move downward from the head toward the feet. The explanation is as follows: The images of objects situated on the subject's left appear on the right side of the retina at successively lower levels, owing to the slow return movement of the fundus with the watch. This is falsely interpreted (projected) as an actual movement of the external objects from below upward. See diagram. Objects situated on the right of the subject cast their images upon the left side of the retina. Owing to the slow movement of the retina with the watch the images appear at successively higher levels on the retina. This is falsely interpreted as an actual movement of external objects from above downward. For similar reasons looking downward in rotary nystagmus against the watch gives the appearance of external objects moving toward the left, whilst looking upward gives the impression of an apparent movement of objects toward the right. It should be remembered, however, that voluntarily turning the eyes strongly in certain directions has a tendency to inhibit or enhance one or other element of the nystagmus. Thus, for instance, looking upward frequently slows or inhibits the rapid element in rotary nystagmus against the watch and at the same time may develop a vertical nystagmus in an upward direction. On account of the location of their insertions in relation to the centre of rotation of the eyeball the superior and inferior oblique muscles tend to rotate the globe downward (Fuchs[100]). Hence, looking up tends to inhibit the play of these muscles in nystagmus and thereby to favor the development of some latent or subsidiary form in another direction.

The general effect of a mixed horizontal and rotary nystagmus to the right and against the watch respectively, is ordinarily the appearance of external objects moving from left to right with a downward slant.

The nystagmus of aural irrigations has already been described. In rotations about the three main axes of the body a characteristic dominant type of nystagmus was found to be constant for the movement in each separate plane. Thus rotation about the long axis from right to left has directly associated with it a primary horizontal nystagmus directed toward the left; rotation in the mesial plane "face first" has directly associated with it vertical nystagmus directed toward the feet, whilst rotation in the mesial plane "occiput first" has directly associated with it vertical nystagmus directed toward the top of the head. In addition to the dominant form of nystagmus, however, there was at times present in the various forms of rotation a subsidiary form. Thus in rotations about the long axis from right to left a rotary nystagmus with the watch was superadded to the primary horizontal nystagmus directed to the left.

The actual movements of the eyes were, of course, best studied in the after-nystagmus, and deductions made from these as to the primary movements as well as from the apparent movement of external objects. The dominant element of nystagmus was also directly observed during actual rotations. In aural irrigations the direction of the elements of the nystagmus was directly ascertained by the aid of the ophthalmoscope and was found to justify the deductions already made in rotations. The cause of the mixed type of nystagmus in rotations was due in part to an irregularity in the direction of the swing whereby the body, instead of revolving strictly about its long axis, described in its course, as a whole, an ellipse, thus approximating a movement to and fro in the coronal plane, thereby affecting the receptors in the ampullæ of the superior canals. In aural irrigations a great factor in the production of the superadded rotary element is to be sought in the anatomical relations of the semicircular canals, whereby the prolonged action of heat or cold affects not only the receptors in the ampulla of the external semicircular canal, but also the receptors in the nearby ampulla of the superior canal, and even those in the macula of the utricle. In rotations in the coronal plane, the chief effect of which is felt in one or other of the ampullæ of the superior semicircular canals, a secondary and subsidiary effect is frequently manifested through the ampullary receptors of the adjacent horizontal canal.

In rotations in the mesial plane the peripheral labyrinthine mechanisms involved are bilateral instead of unilateral as in rotations in the coronal plane or about the long axis. Thus in rotations in the mesial plane "occiput first" the ampullæ of both posterior canals are chiefly affected, whilst their natural opponents in rotations in the mesial plane, viz., the ampullæ of both superior canals are chiefly affected in rotations "face first." In rotations in the mesial plane the nystagmus is almost purely vertical being primarily directed toward the feet in rotations "face first" and toward the vertex capitis in rotations "occiput first." If the rotations be confined strictly to the sagittal plane no horizontal or rotary element will appear in normal individuals, because under the conditions supposed the ampullæ of each pair of canals are equally affected, for in mesial rotations the ampulla of one canal upon one side is not opposed functionally to its fellow of the opposite side, as in coronal and horizontal rotations, but both ampullæ of the superior canals are the direct opponents of both ampullæ of the posterior canals. Since, therefore, horizontal and rotary nystagmus are caused respectively by unilateral affection of a horizontal or a superior canal respectively, it is clear that in mesial rotations these elements are generally absent. Here, again, aural irrigations have come to the rescue, for whilst horizontal and rotary nystagmus occur simultaneously in one-sided hot and cold irrigations, when such irrigations were given bilaterally and with care, the only nystagmus produced was of the vertical type. In the case of bilateral cold irrigations the vertical nystagmus was directed toward the head, as directly observed by means of the ophthalmoscope. As this form of nystagmus is the same as that which directly accompanies rotations in the mesial plane "occiput first" and as bilateral cold irrigations act by depressing equally the receptors in the ampullæ of the superior and horizontal canals of both sides which, as we have just seen, has no effect or a neutral one in causing horizontal and rotary nystagmus, it is evident that the vertical nystagmus toward the vertex must result from a disturbance of the mechanisms involved in mesial rotations, viz., by means of absolute depression of irritability (increased threshold value) of the receptors in the ampullæ of the superior canals or, which amounts to the same thing, a relative enhancement of irritability (lowered threshold value) in the receptors of the ampullæ of the posterior canals.

In bilateral hot irrigations the vertical nystagmus is directed toward the feet. In these irrigations the peripheral mechanisms involved in mesial rotations were disturbed by enhancement of irritability (lowered threshold value) in the receptors of the ampullæ of the superior canals, resulting in nystagmus that is identical in nature and direction with that which is directly associated with mesial rotations "face first." In this way, by means of aural irrigations, a knowledge of the function of the posterior canals has been obtained, even though they are too deeply situated to be directly affected by heat or cold applied within the external auditory canal. And this knowledge accords in every respect with what has been determined by animal experimentation. Ewald, [76] by means of his pneumatic hammer, and Breuer, [75] by means of suction and insufflation, found, on forcing the endolymph toward the ampulla of the horizontal canal, a stimulus reaction in which the head went to the opposite side and on withdrawal of the hammer a weaker reaction of retardation caused by the current of endolymph from the ampulla toward the canal. The stimulation effect obtained thus by Ewald from a current of endolymph toward the ampulla corresponds exactly with those effects obtained from hot irrigations on one side or directly from rotation about the long axis toward the side in which the canal under observation is located. Ewald's experiment shows conclusively that in rotations about the long axis the chief effect upon the ampullary nerve endings is brought about by whatever tends to create a current of endolymph toward the ampulla from the canal, and that a secondary retardation effect, much less in degree, is to be expected from whatever tends to create a current of endolymph from the ampulla toward the canal. Owing to the fact that the calibre of the membranous canals is capillary in size, an actual movement of the endolymph from the ampulla toward the canal is scarcely possible in rotations calculated to cause a flow of endolymph in that direction. An alteration of pressure conditions, if not an actual movement of endolymph, is sufficient to stimulate the ampullary receptors so as to upset the delicate balance of the labyrinthine tonus apparatus. The delicacy of the ampullary receptors is extreme. All sorts of stimuli seem to affect them, e.g., galvanic, tactual, thermal, and even photo stimuli.

Looking now to the location and direction of the semicircular canals and their relations to each other it is evident that in a rotation about the long axis, let us say from left to right, there is created in the right horizontal canal a tendency to drive the endolymph toward the ampulla from the canal, and if the subject's head be tilted backward at the same time, there is created in the left superior canal a tendency for the endolymph to flow from the ampulla toward the canal. In rotations in the mesial plane "occiput first" there is a tendency to drive the endolymph away from the ampullæ of both posterior canals, causing vertical nystagmus toward the vertex. In mesial rotations "face first" there is a tendency to drive the endolymph away from the ampullæ of both superior canals with the production of vertical nystagmus toward the feet. The absence of rotary nystagmus in these mesial rotations is accounted for by the simultaneous and equal affection of the opposing sets of receptors upon either side of the body. The views here expressed have the support of Ewald,[78] who found that whereas a flow of endolymph toward the ampulla from the canal caused the stimulus reaction in the horizontal canal, the opposite was true, for the superior and posterior canals in which the flow from the ampullæ toward the canals had the primary stimulating effect, whilst a flow toward the ampullæ caused the weaker reaction of retardation.

In rotations against the watch in the coronal plane with the subject lying supine, the mixed after-nystagmus was composed of a rotary element with the watch and a vertical element directed toward the feet. External objects seemed to move from the head toward the feet in a slanting direction, roughly indicated by a line drawn from the left eye to the right great toe. By stopping the swing suddenly, a current of endolymph, away from the ampulla, was created in the left superior canal, thereby causing the after-nystagmus just described. The apparent movement of objects in this instance evidently resulted from enhancement of the downward vertical element due to the peculiarities of insertion of the superior and inferior oblique muscles. This element is normally latent in pure rotary nystagmus, but may be developed by looking toward the feet.

Whether the tendency to a reflux toward the canal from the ampulla, or vice versa, is important in determining the secondary nystagmus and other effects, seems doubtful, since the conditions in rotations as the swing slows down, can hardly be compared with those brought about by Breuer's suction or by stripping the membranous canals by means of the Ewald hammer. At all events the tendency to positive counterpressures, created in the canals of the opposite side by slowing or stopping of the swing, overshadows such retardation effects.

CHAPTER XVIII

THE EFFECTS OF THE GALVANIC CURRENT UPON THE SEMICIRCULAR CANALS

Numerous observations were made on various subjects and with the anode and cathode in various positions, such, for instance, as with one electrode over the mastoid area whilst the other was held in the hand of the same or opposite side, or fixed upon the breast. All sorts of combinations were tried. The electrode applied to the mastoid area was held in position by a bandage the better to appreciate the smaller degrees of reactionary movements of the head.

The general results were as follows: With the positive pole over the left and the negative over the right mastoid area, the head went strongly to the left side on closure of the circuit. The eyes were at first drawn to the left side but later showed mixed nystagmus, the horizontal element being directed to the right and the rotary element directed against the watch. External objects seemed to move from left to right and downward. The retinal vessels were at first dilated and later contracted. There were vertigo, pallor, nausea, "lump-sensation" in the stomach, increased saliva, etc. In fact the phenomena were in every way analogous to those seen with cold irrigations of the left ear.

On opening the circuit the head was immediately pulled toward the right, i.e., into the vertical position. With the anode over the left mastoid and the cathode over the left breast similar phenomena were observed, but of less degree. With the cathode over the left mastoid area and the anode over the left breast the head was drawn to the right on closure of the circuit, and the eyes showed mixed nystagmus, the horizontal element being toward the left, whilst the rotary element was with the watch. These movements of the head and eyes were of greater degree than the similar movements noted with the positive pole over the left mastoid. The reaction of the cathode over the left mastoid area was in every respect analogous to a hot irrigation of the left ear.

During the continued flow of the current, if the subject closed his eyes he felt his body revolving about its long axis in the same direction as that taken by the horizontal element of the nystagmus. Let us suppose the case where the anode is over the left mastoid and the head inclines to the left, whilst the eyes are in nystagmus with the horizontal element toward the right. If the subject close his eyes he will imagine himself revolving about his long axis from left to right. This is exactly what the subject experiences sitting upright with closed eyes during a cold irrigation of the left ear. It is also the counterpart of what occurs upon sudden slowing or stopping of a rotation about the long axis from right to left.

A male subject, aged 55, afflicted with tabes dorsalis, but with normal hearing, showed the usual reaction to galvanism. On standing up the disturbances of equilibrium on opening and closing the circuit were very marked. This subject seemed more susceptible to galvanic vertigo than the usual run of people. After the application, which was very mild and did not sicken or distress him as did irrigations practised on a previous occasion, it was noted that for a time the subject walked and balanced better than before the application.

In a normal person the anode and cathode were carefully secured over the left and right mastoid areas respectively, and careful note was made of the degree of response made on closure of the circuit as measured by the deviation of the head and body.

A bilateral aural irrigation at 121° F. was then given, with the electrodes still *in situ* and immediately after the irrigation the circuit was closed. It was found that after the hot irrigation the reaction was markedly increased, the head and body going much more strongly toward the anode. In addition the susceptibility of the subject was otherwise enhanced, for the current, after the irrigation, made him feel nauseated and much sicker and dizzier than it had ever done before, so that he became very pale, weak and tremulous. Care was taken to use currents of equal strength in each case and to eliminate every possible source of error. It was noted that the increased irritability to galvanism of the semicircular canals soon disappeared.

With the cathode over the left and the anode over the right mastoid area, tests were carefully made before and after irrigation of the left ear at 115° F. It was noted that after the irrigation there was markedly increased deviation of the head to the right on closure of the circuit. The hot irrigation also caused gradually increasing pain in the vicinity of the drum membrane until the flow of water became unbearable.

Similar tests were made with the cathode over the left mastoid and the anode over the left breast. It was noted that after irrigation of the left ear at 115° F. the deviation to the right was increased on closure of the circuit compared with that noted before the irrigation. With the cathode still over the left mastoid it was found, after irrigation of the right ear with water at 115° F. that deviation to the right was diminished or absent on closure of the circuit. Currents of equal strength were used before and after the irrigation.

Similar tests were made with the anode over the left mastoid and the cathode over the left breast. After irrigation of the left ear at 115° F. there was no deviation on closure of the circuit. Immediately the position of the poles was reversed so that the cathode was placed over the left mastoid and then closure of the circuit resulted in a marked deviation to the right. It was noted that if these tests were made with the subject standing up, the disturbance of equilibrium was much more manifest and the subject's sickness, nausea, etc., more pronounced than when the tests were made with the subject sitting erect. After the last hot irrigation the subject deviated to the right in walking with the eyes closed.

Galvanism was tried on a man 25 years old in whom there was practically no drum membrane in the left ear, whilst there were two fair-sized perforations in the right drum membrane. It was at once apparent that this man was unusually susceptible to the effects of galvanism as compared with all the other subjects examined. It should be mentioned, however, that previous to the application of the electrodes he had had cold and hot irrigations, the last one being at 116° F. With the anode over the left, and the cathode over the right mastoid area and properly secured, on closure of the circuit the head inclined to the left. The eyes were at first drawn to the left and were rotated strongly in the direction of the hands of the watch. It was soon apparent, however, that a mixed nystagmus was present, with the horizontal element directed to the right, and the rotary element, which was unusually well marked, directed against the watch. During the continuance of the current this nystagmus persisted to the right and against the watch. During the continued flow of the current on turning the eyes strongly to the left the rapid jerks of the rotary nystagmus seemed to be lengthened and slowed, and external objects seemed to move up and down. On looking up there seemed to be less motion of the eyes, and what motion there was took place in the horizontal plane to the right. External objects seemed to move in the same plane and direction. On looking down, the rotary element was still against the watch, and objects seemed to jump from side to side. On looking to the right the eyes jerked to the right and against the watch. On looking to the left moderately, the eyes jerked mainly against the watch, and objects appeared to go upward on the left.

At the moment of "making" with the eyes directed to the right, the eyeballs were drawn to the left and rotated with a steady pull in the direction of the hands of the watch. On looking to the left at the moment of "making" the eyes were rotated strongly with the watch. On looking up and on looking down, the eyes were similarly drawn to the left and rotated with the watch at the moment of closure of the circuit. With the anode over the right, and the cathode over the left mastoid area, similar phenomena were noted. The head inclined to the right. There was mixed nystagmus with the horizontal element to the left and the rotary element with the watch. Objects seemed to pass around the subject's body in the direction of the hands of the watch in the coronal plane.

The conclusions are:

1. That the effects of closure of the circuit with the

anode over the left mastoid are exactly like those of cold irrigations of the left ear which, in turn, are like the primary effects of rotation about the long axis from left to right or the secondary effects of a similar rotation from right to left.

2. That the effects of closure of the circuit with the cathode over the left mastoid area are identical with those of hot irrigations in the left ear, and with the primary effects of rotation from right to left about the long axis or the secondary effects of a similar rotation from left to right.

3. That on closure of the circuit with the anode over the left, and the cathode over the right mastoid area, the effect is enhanced, just as in the case of simultaneous irrigation of the two ears with water at 79° F. in one ear, and 107° F. in the other, after it had been shown that single irrigations at either one of these temperatures was without manifest effect.

4. That hot irrigations affect the labyrinthine receptors in such a way that with the cathode over the mastoid area on the side of the irrigation the usual effect is enhanced, whilst with the anode over the mastoid of the side of the irrigation no effect is manifested or experienced by the subject.

5. That, as a corollary to No. 4, hot irrigations act by increasing the irritability of the homolateral ampullary receptors, just as cold irrigations act by depressing them.

CHAPTER XIX

HOW ROTATIONS, AURAL IRRIGATIONS, AND GALVANISM AFFECT THE LABYRINTHINE RECEPTORS AND THE RELATED EFFECTORS

It has already been shown that in rotations the chief effect upon the ampullary receptors is caused by changes of pressure conditions resulting from a tendency of the endolymph to flow in a particular direction, e.g., toward the ampulla in the case of the horizontal canals, and away from the ampulla in the case of the superior and posterior canals. The experiments of Ewald[76] and of Breuer,[75] by stripping, insufflation and suction of the membranous canals, were cited in this connection. Furthermore it has been shown that, with the subject in the erect posture, labyrinthine balanced mechanisms exert, constantly, a reflex tonus upon part at least of the muscles involved in equilibration and that enhancement or depreciation of the irritabilty of the labyrinthine mechanisms, or of part of them, upon one side of the body caused disturbances of equilibrium and nystagmus, both of which presented a constant characteristic type depending upon the part (canals) affected. It has, moreover, been shown that cold irrigations act by depressing the normal irritability of the ampullary and perhaps the macular receptors, whereby the tonus exerted reflexly through the related nervous arcs is diminished on one side, thus upsetting the reflex balanced mechanisms with consequent deviation of the head toward the side of irrigation and horizontal nystagmus directed toward the opposite side. On the other hand, hot irrigations act by increasing the irritability of the labyrinthine receptors affected, thereby disturbing the reflex balanced mechanisms with deviation of the head to the opposite side and horizontal nystagmus directed to the side of irrigation The enhanced or depressed irritability of the receptors acts by increasing or diminishing the quantitative value of the afferent impulses normally generated by movements of the head, rotations, etc. That this is so the following experiment seems to prove. The subject sitting erect was rotated about the long axis from left to right. The after-nystagmus of mixed type was directed to the left and with the watch. An irrigation of the left ear for 4 minutes at 68° F. was now given, and immediately the rotation from left to right was repeated. On suddenly arresting the motion there was found to be no after-nystagmus, either of the horizontal or rotary type toward the left or with the watch. On looking to the left, the eyes were steady, with a tendency to rotate rather slowly against the watch. With the eyes held straight or turned to the right, there was a violent mixed nystagmus with the horizontal element directed to the right and the rotary element against the watch. In other words, instead of the usual after-nystagmus appearing, the primary nystagmus persisted, and traces of it were found for over two minutes after the sudden arrest of the rotation. This also shows conclusively that, in the sudden reversal or slowing of rotations about the long axis, the active factor in causing after-nystagmus and the associated phenomena, is not the tendency to reflux on the part of the endolymph from the ampulla toward the canal upon the side toward which the rotation had been directed, i.e., in this case on the right side but a positive tendency, created by the sudden cessation of the rotation, in the opposite labyrinth of the endolymph to flow from the canal toward the ampulla. When the cold irrigation in the left ear had deadened the irritability of the ampullary receptors of the left horizontal and superior canals the pressure conditions created by the tendency of the endolymph to flow toward or away from the ampullæ were without effect. In this manner the usual after-nystagmus of the rotation was suppressed, and the primary nystagmus allowed to persist, even for some minutes after the rotation had been suddenly checked.

At a later period this subject was more fully studied. Rotations in various planes were given immediately after unilateral and bilateral hot and cold irrigations. Because of the help they afford in determining the mechanism of labyrinthine phenomena the results are deemed of sufficient importance to warrant free transcription of the notes taken during the experiments.

With the subject sitting upright in the swing, bilateral aural irrigation at 54° F. caused no nystagmus and no apparent movement of external objects. Immediately after the irrigation a brisk rotation was given about the long axis from right to left. Upon sudden arrest of the swing there was no nystagmus and no apparent movement of external objects, but the subject belched violently and vomited mucus. During the rotation there was a slight feeling of sickness and the subject could see external objects passing before his eyes, but visual fixation was impossible. It was noticeable that when the rotation ceased the subject experienced no dizziness or distress in the head, did not stagger or deviate in walking, and did not feel weak or want to lie down, whilst the external world seemed steady and "solid as a rock." All these things occurred in spite of the fact that stopping the swing caused violent vomiting. The subject, however, looked very pale from constriction of the skin-vessels. After a rest of 25 minutes the pulse-rate and blood-pressure were 56 and 115 respectively. The knee-jerks were fairly active. Bilateral irrigation at 54° F. caused, just after its commencement, the blood-pressure to rise to 130, the pulse being 72. After two minutes of irrigation the pulse-rate and blood-pressure were still at 72 and 130 respectively. There was no nystagmus during the irrigation, though the patient felt a little sick in the stomach. Immediately following the irrigation a hard swing was given about the long axis, from left to right. Abrupt stopping of the swing did not cause distress in the head or nystagmus. There was no apparent movement of external objects, but again the subject belched violently and vomited bile-stained mucus. Immediately on stopping the rotation the pulse-rate and blood-pressure were 60 and 135 respectively. This was before the vomiting had occurred. After vomiting, i.e., about one minute after the rotation, the pulse-rate and blood-pressure were 80 and 140, and 80 and 130, whilst the knee-jerks were about the same as before irrigation. Five minutes after the rotation the pulse-rate and blood-pressure were 64 and 115. During unilateral irrigation in left ear at 54° F. the pulse-rate and blood-pressure were 64 and

125, and there was present an irregular vertical nystagmus, with an apparent up and down movement of objects. At the onset of the irrigation the subject felt sick. Just before the onset of vomiting there was audible gurgling in the stomach. At this moment the pulse-rate and blood-pressure were 76 and 125. After vomiting, the pulse-rate and blood-pressure were 84 and 145. The knee-jerks were slightly affected, the right being perceptibly diminished. The absence of horizontal nystagmus in this irrigation was due to the fact that the ampullary receptors in the horizontal canals had not recovered from the effects of the previous irrigations (exhaustion). The irregular vertical nystagmus was due to a rotary element caused by the effects of the irrigation upon the ampulla of the left superior canal. It was noticed that with the unilateral irrigation the subject broke into a sweat and felt very weak. These symptoms were absent in bilateral irrigations.

After a rest the subject was drowsy. The pulse-rate and blood-pressure were 56 and 110. The left knee-jerk was somewhat more active than the right. During bilateral irrigation at 115° F. there was slight vertical nystagmus. The subject felt dizzy and sick, and external objects seemed to move from head to foot and a little toward the right. The pulse-rate and blood-pressure were 64 and 130. Rotations about the long axis from left to right and from right to left were rapidly given after the irrigation. Upon checking the rotation from left to right there was horizontal after-nystagmus to the left. Similarly, upon checking the rotation from right to left, there was horizontal after-nystagmus to the right. Each rotation made the subject very dizzy, sick, and weak, but there was not so much tendency to vomit as after the previous rotations. This was due in part to the fact that the stomach had been so recently emptied. After the last rotation, which was from left to right, the subject felt very distressed in the head, and external objects moved rapidly "in a flurry" without any particular direction. There was no sweating or vomiting. The knee-jerks were not altered, the left being still slightly stronger than the right. The respiration was heavy, the subject complaining that he felt as if a weight were on his chest. Immediately following the first swing, the pulse-rate and blood-pressure were 64 and 120. Five minutes after the last rotation the subject felt "done up." His eyes were tired and felt like closing. The pulse-rate and blood-pressure were 64 and 115.

On another occasion in the resting state the pulse-rate and blood-pressure were 60 and 115. During irrigation of the left ear at 52° F. the usual horizontal nystagmus to the right was evident. When the eyes were strongly turned to the left, there were jerky rotary movements against the watch. With the eyes open and turned to the left, the subject felt his body turning on its long axis from left to right. With the eyes open and turned to the right, the subject felt his body rotating to the right, but not so much so as when his eyes were turned to the left. This unusual phenomenon occurred during irrigations practised with the subject sitting in the swing with the feet off the floor, and is interesting because it shows to what an extent we rely upon other senses for corroboration of labyrinthine impressions in estimating our relations in space. When the subject sat upon a chair with the feet upon the floor and with the eyes open and turned to the left, no sense of motion was experienced in similar irrigations. During a swing from right to left, immediately following irrigation of the left ear at 52° F. the subject saw external objects as a streak. Visual fixation was impossible. The tonus required for the slow element of the primary nystagmus was wanting. The after-nystagmus to the right was violent, the eyes oscillating back and forth with excursions of equal length and duration, and highly suggestive of the nystagmus of gross cerebellar defect. Later the eyes steadied down somewhat and showed slight horizontal nystagmus to the right. At first during the after-nystagmus, external objects seemed to move back and forth furiously from side to side. During the rotation the subject could hardly think, and the head inclined to the left shoulder instead of to the right, as is usual in rotations from right to left. There was no dizziness, and external objects went around from left to right. Upon cessation of the rotation the head still inclined to the left shoulder and the subject felt sick. The inclination of the head toward the left shoulder during the rotations is a further proof that deviations of the head in rotations, aural irrigations and galvanism, are the direct result of changes of the labyrinthine tonus in the muscles of the head and neck. It also shows that in the horizontal canals, stress in the endolymph directed away from the ampulla is not a factor in the compensating movements of equilibration. If it were such a factor, then during the rotation from right to left, when the receptors in the ampulla of the left horizontal canal had previously been deadened by cold irrigation, we should expect that the stress in the endolymph of the right horizontal canal away from the ampulla would draw the head toward the right shoulder. The evidence pointed merely to relatively increased tonus of the right labyrinth caused by depression of the receptors in the left labyrinth. To this and this alone, must be attributed the deviation of the head.

Irrigation of the left ear at 50° F. given 10 minutes after the previous irrigation, caused distress in the stomach rather than in the eyes or head. There was little or no nystagmus even in turning the eyes to one side or the other. The head fell toward the left and visual fixation was possible. After the irrigation a hard swing from left to right was given. During the swing the subject vomited, and the head fell to the left but visual fixation was possible. On cessation of the swing the head still fell toward the left. There was no after-nystagmus on looking straight ahead or to the right. There was a slight tendency to horizontal nystagmus on looking toward the left. From this it seems that the tonus innervation associated with the slow elements is the important factor in initiating nystagmus. Thus, upon cessation of the rotation, stress of the endolymph in the left horizontal canal was created toward the ampulla, but owing to the depressed condition of the receptors (increased threshold value) no tonus effect was manifested in the muscles on the right side of the head or in the muscles moving the eyeballs to the right. As a result the head still fell toward the left, and no after-nystagmus developed. The absence of nystagmus during the last cold irrigation seemed due to the fact that compensatory adjustment for the disturbed labyrinthine relations had already been made through ocular impressions, aided by voluntary effort. Moreover, the condition of the receptors in the left labyrinth, after so many cold irrigations, was such that effects from further cold irrigations were temporarily absent.

With the subject lying supine in the swing and resting, the pulse-rate and blood-pressure were 48 and 125. During bilateral irrigation at 50° F. there was no nystagmus or discomfort, whilst the pulse-rate and blood-pressure were 48 and 135. Immediately after the irrigation a brisk rotation was given in the coronal plane and with the watch. On suddenly checking the swing the usual after-nystagmus was present, i.e., rotary against the watch with external objects appearing to move about the subject's body, i.e., upon the left, over the head and down the right side. The nystagmus lasted only a moment or two. From this it seems that bilateral cold irrigations do not completely abolish the function of the ampullary receptors in the superior canals.

After bilateral irrigation at 50° F. rotation in the sagittal plane "occiput first" caused vertical after-nystagmus toward the feet and with a slight lateral tendency toward the right. During the swing visual fixation was impossible and the subject was made very dizzy.

Rotation in the sagittal plane "face first," given after bilateral irrigation at 50° F., caused vertical after-nystagmus with up and down movements of the eyes. The direction of the movements was slanting from the left side of the head toward the right foot. External objects seemed to move "up and down" in the same direction. The abrupt stopping of this last rotation made the subject very sick. This result, unusual for such rotations, is to be attributed to the relatively enhanced irritability of the ampullary receptors of the posterior canals caused by the depression of the ampullary receptors of the superior canals following the irrigation. The irrigations given in these experiments all lasted not less than 3, nor more than 4 minutes.

The effect of galvanism upon the semicircular canals resolves itself also into one of exaltation or depression of the ampullary receptors. The well-known phenomena of electrotonus, coupled with the foregoing observations, afford an easy explanation. Thus, with the cathode over the left mastoid area the adjacent ampullary nerve elements are put in the state of katelectrotonus, i.e., their irritability is increased as in the case of hot irrigations in the left ear, and there is lateral inclination of the head toward the right with horizontal nystagmus toward the left. With the anode over the left mastoid area, the adjacent ampullary nerve elements are put in a state of anelectrotonus, i.e., of decreased irritability with resulting phenomena similar to those of cold irrigations in the left ear.[149] With the cathode over one mastoid area, and the anode over the other, the maximum effect is produced as when a cold irrigation is given in one ear and a hot irrigation in the other simultaneously.

From the definite direction of the movements that occur in response to rotations, aural irrigations and galvanism, it is manifest that the receptors of each semicircular canal and vestibule are functionally related to definite groups of muscles. Thus in hot irrigations of the left ear, the head is actively drawn toward the right shoulder, and since this is the result of temporarily increased irritability (lowered threshold value) of the ampullary receptors in the left horizontal canal, it is evident that these receptors and their associated arcs are in relation with the muscles on the right side of the head and neck which govern such a movement. Similarly, in cold irrigations of the left ear by depression of the ampullary receptors of the left horizontal canal and consequent diminished tonus in the muscles functionally connected with them, viz.: those of the opposite side of the neck and head, there is deviation of the head toward the left. Thus it is manifest that a crossed relation exists between the horizontal canal on one side of the body and the muscles with which it is functionally associated upon the other side. When the muscular associations of the ampullary receptors of the superior and posterior canals are considered, the evidence points to the fact that each pair of canals acts as the antagonist of the other pair when both members of one or other of the pairs are equally and simultaneously stimulated or depressed, as in the case of bilateral hot or cold irrigations, or in rotations in the sagittal plane. The evidence obtained from various sources points to the fact that the receptors of the superior canals are associated functionally with the muscles of the head and neck, situated dorsally, which tend to draw the head backward. The evidence of this relation is supplied in part by the phenomena manifested during bilateral cold irrigations where the head falls forward from depreciation of the normal reflex tonus in the muscles referred to. Furthermore the receptors of each superior canal seem to be in relation with the muscles of the head and neck of the opposite side and situated dorsally, i.e., they are in crossed relation with one-half of the muscles just mentioned as tending to draw the head, when bent forward, back into the vertical position. This conclusion is based upon the fact that in single cold irrigations, say of the left auditory canal, the head inclines not only to the left, but rotates somewhat on the long axis of the body, so that the chin points toward the right. Similarly it may be deduced that the posterior canals are associated functionally with the muscles of the neck which tend to draw the head forward. This rough general outline of the muscular associations of the ampullary receptors, based on clinical studies, is in harmony with the phenomena that follow section of the canals in animals.

There are thus efficient balanced mechanisms constantly in action, whereby every displacement of the head is compensated or corrected in a reflex manner. The influence of the otoliths is intentionally omitted to avoid confusion. Normally, therefore, equilibrium is maintained reflexly, each displacement being met by active stimulation (enhanced tonus) of the muscles tending to correct such displacement or by inhibition (diminished tonus) of those muscles that tend to produce it.

In studying the muscles involved in the mechanism of nystagmus caused by rotation, aural irrigations, or galvanism, one is struck at once by the similarity of their behaviour to that of the muscles that control the displacements of the head. Thus in displacements of the head in aural irrigations, the muscles upon the side toward which the displacement takes place are in an absolutely or relatively heightened state of tonus whilst their opponents of the opposite side are in a state of diminished tonus. Similarly the muscles that tend to turn eyeballs in one direction or the other are for the moment in a heightened state of tonus, whilst the tonus of their opponents is momentarily depressed. This rule applies to the rotary and vertical as well as to the horizontal movements of the eyes. The grouping of the opponent muscles in nystagmus corroborates what has been stated concerning the grouping of the muscles controlling displacements of the head. Thus the external rectus of one eye works in conjunction with the internal rectus of the other eye, both being in turn opposed by the analogous muscles on the opposite side of each eyeball. In vertical nystagmus both inferior recti are the opponents of both superior recti. In rotary nystagmus the superior oblique of the right eye, and the inferior oblique of the left, act in conjunction, their opponents being the inferior oblique of the right and superior oblique of the left eye respectively. In nystagmus, the long steady pull of the eyes indicates in a general way that the muscles responsible for it are in a state of heightened tonus (absolute or relative), whilst the short jerky movements that give direction to the nystagmus indicate in general a lessened tonus or rhythmic inhibition in the related muscles. Thus in cold irrigations of the left ear with displacement of the head toward the left, and horizontal nystagmus toward the right, the steady pull of the eyeballs to the left indicates that the muscles responsible for it are in a relatively heightened state of tonus, and are consequently in functional relation with the ampullary receptors of the right horizontal canal which are relatively exalted as to their sensibility (lowered threshold value), whilst their opponents are in relation with the ampullary receptors of the left horizontal canal which are absolutely depressed by the action of the cold irrigation. The tonus in the muscles associated with either set of movements is not constant, but is subject to alternating changes effected by means of reciprocal innervation.

Again, in rotations in the mesial plane, let us say "occiput first," the primary nystagmus is directed toward the vertex capitis, with the slow and steady movement, during which vision is possible, directed toward the feet. The latter movement shows that the apparatus responsible for it is in a heightened state of tonus corresponding with the enhanced irritability of the ampullary receptors of both posterior canals. Similarly, in rotations in the mesial plane, "face first," the steady pull of the primary nystagmus is toward the vertex corresponding with the heightened irritability of the ampullary receptors in both superior canals.

In rotary nystagmus the superior oblique acts under the influence of the ampullary receptors of the superior canal of the same side, and perhaps of the posterior canal of the opposite side. Owing to the decussation of the fourth nerve the relation between the superior and posterior canals and the superior oblique muscle is an uncrossed one. See diagram. A glance at a good specimen of the bony canals exposed *in situ*, with the relations to the various parts and planes of the skull preserved, will at once convince one that rotation in the coronal plane

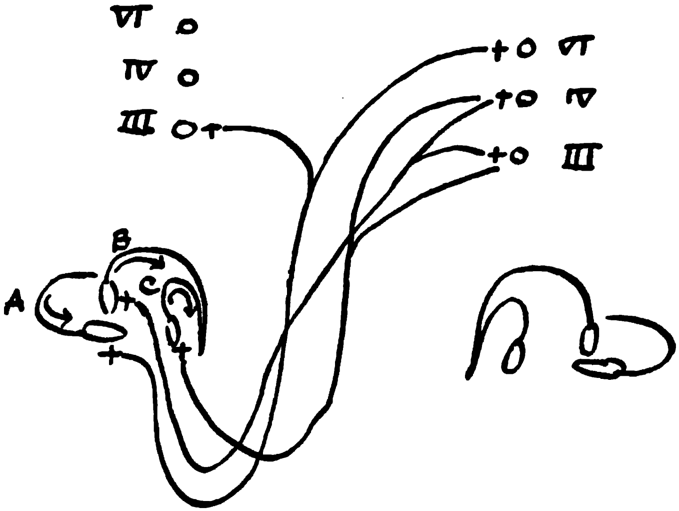


Diagram illustrating the relations of the semicircular canals to the oculo-motor nuclei. A, the right horizontal canal; B, the right superior canal; C, the right posterior canal. The Roman numerals indicate the oculo-motor nuclei. The arrows indicate the direction in which the tendency of the current of endolymph begets the stronger, irritative reaction.

against the watch tends to displace the endolymph in the right superior canal from the ampulla toward the canal.

Similarly, the position and direction of the superior canals is such that the endolymph in them is liable to be influenced by aural irrigations, but not by rotations strictly confined to a plane perpendicular to the long axis. In rotations in the coronal plane with the watch, there is a tendency to displacement of the endolymph away from the ampulla in the left superior canal. The ampullary receptors of the left superior canal are thus actively stimulated, thereby enhancing the tonus of the related muscles, viz., the inferior oblique on the right, and the superior oblique on the left side, which initiates rotary nystagmus with the watch.

In unilateral cold irrigations the posterior canal is not affected, unless perhaps the irrigation is very prolonged, and then the effect is to reinforce that of the superior canal of the opposite side, since these canals stand in the relation of antagonists probably.

Summary of the Relations of the Ocular Muscles.

1. The external rectus is mainly in relation with the ampullary receptors of the horizontal semicircular canal of the opposite side.
2. The superior oblique is mainly in relation with the ampullary receptors of the superior and possibly with those of the inferior canal of the same side.
3. The internal rectus is mainly in relation with the ampullary receptors of the horizontal canal of the same side, though the part of the third nerve nucleus governing its associated movement with the external rectus may, on account of the partial decussation within the nucleus be in relation with the horizontal canal of the opposite side, i.e., the muscle and the origin of its motor neurones are on opposite sides of the median line.
4. The inferior oblique is mainly in relation with the ampullary receptors of the superior and posterior canals of the opposite side.
5. The superior and inferior recti, as well as the levatores palpebrarum and part of the orbiculares palpebrarum, act synchronously on both sides of the body, and are

therefore in relation with the receptors of the semicircular canals of both sides, the superior recti and the levatores palpebrarum being in relation with the superior semicircular canals, whilst the inferior recti and the orbiculares are in relation with the posterior canals. The relations between the nuclei of the III, VII, and XII nerves have already been discussed. These bilateral connections are affected through the median portion of the third nerve nucleus or through the partial decussation that occurs within the nucleus, since some anatomists (Tsuchida[14]) have recently denied the existence of a well-marked unpaired central nucleus as described by Perlia and apparently sustained by the observations of Starr and those of Kahler and Pick.[131]

It should be noted that the superior oblique of one eye is associated with the inferior oblique of the opposite eye in the mechanism of rotary nystagmus.

The associations of the ocular muscles above enumerated are based on numerous observations of the nystagmic movements that accompany rotations, aural irrigations and galvanism. In the light of these associations the phenomena that follow section of the vestibular nerve, in the sheep for instance, as done by Biehl,[70] are readily explained. Thus, in section of the right vestibular nerve the right eye was turned downward and outward, whilst the left was turned upward and inward. Here the internal rectus in the right and the external rectus in the left eye were severed from their labyrinthine receptors through which they derived their reflex tonus, thus relatively enhancing the tonus of their opponents with a tendency of the eyes to deviate in general toward the right. Again the right superior oblique and left inferior oblique are severed from their labyrinthine receptors with consequent loss of reflex tonus which relatively enhances the tonus of their opponents with a tendency of the right eye to be drawn downward as a whole, by the right inferior oblique and a tendency of the left eye to be drawn upward as a whole by the unopposed action of the left superior oblique. The superior and inferior recti, as well as the levatores and orbiculares palpebrarum, are so affected that no deviation results owing to the fact that antagonistic muscles are equally affected, and that these muscles are in free relation with the semicircular canals of both sides. Peculiarities in regard to the site of insertion of the ocular muscles, and more especially of the oblique muscles and difficulty in appreciating the net mechanical result of the working action of the ocular muscles, as a whole, have been the cause of much discrepancy in the nystagmus and deviations observed after section of the VIII nerve and in other experiments upon the labyrinth and cerebellum.

Attempts to study the deviation of the eyes under direct irritation of each separate canal were made by De Cyon,[67] Högyes,[215] Lee[216] and others, but owing to the twofold element of the nystagmus, coupled, in some instances, with the difficulty of confining the effects of irritation to one canal, the results were conflicting. Moreover, the labyrinthine receptors are exceedingly sensitive to thermic and other forms of stimuli, responding even to light (Schwartze[217]). De Cyon himself revised and corrected his first findings, but his later findings, according to Ferrier,[8] were even more unsatisfactory than his first ones.

We have seen that whatever tends to produce a current of endolymph toward the ampulla of the horizontal canal and away from the ampullæ of the posterior and superior canals, causes characteristic primary phenomena by way of displacement of the head in a certain direction with nystagmus of a certain type and direction, whilst in the experiments of Ewald,[76] secondary phenomena of retardation accompanied manipulations tending to drive the endolymph in the reverse direction. With due credit to Lee,[216] who showed that the compensatory movements occurring in fishes subjected to rotation exactly correspond with those caused by direct irritation of the canals, the results of experiments such as those done by Högyes on animals, and repeated by surgeons on the human subject are always open to suspicion, for touching the horizontal membranous canals may give directly opposite results, depending on the direction in which the endolymph is forced. Experiments like these are the chief cause of the interminable discussions upon the symptomatology of labyrinthine disease as to whether the phenomena are due to irritation or destruction. The only rational way to analyze such symptoms is to consider them with reference to enhanced or depressed irritability (absolute or relative) of the labyrinthine receptors on either side of the body and in each of the three semicircular canals. With such characteristic and definite phenomena as the normal reactions afford, little difficulty should be experienced in the interpretation of labyrinthine reactions in diseased conditions.

CHAPTER XX

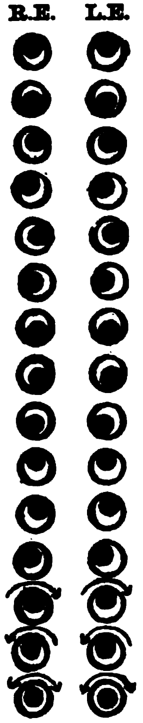
MECHANISM OF THE NYSTAGMUS OF ROTATIONS, AURAL IRRIGATIONS AND GALVANISM

The crossed relations of the semicircular canals to the ocular muscles (excepting the superior oblique) and to the muscles that control the movements of the head have been fully discussed. It remains to consider the relations of the nuclei of the nerves supplying these muscles with motor influence. For the sake of simplicity the ocular muscles alone shall be discussed in this respect, but the deductions will apply equally to all other muscles that receive tonus through afferent labyrinthine impulses. Since Duval and Laborde[80] found that irritation of the sixth nucleus caused conjugate deviation of the eyes toward the side of stimulation, it was believed that the sixth nucleus co-ordinated and controlled the action of the external rectus of the same side and of the internal rectus of the opposite side. This necessitated a connecting link between the sixth nucleus of one side and that part of the third nucleus controlling the action of the internal rectus of the opposite side. The posterior longitudinal fasciculus afforded an easy way of communication, and it was assumed that fibres passed from the sixth nucleus by way of the posterior longitudinal fasciculus to the nucleus of the third nerve of the opposite side. Schäfer,[11] however, denies that any fibres pass from the VI or III nucleus to the posterior longitudinal fasciculus. A little consideration suffices to convince one that if the sixth nucleus controls the conjugate deviation of the eyes, some other nucleus must control the combined action of the muscles that cause the characteristic displacement of the head which accompanies the conjugate deviation of the eyes. Still further consideration will convince that yet another centre must be assumed to control the movements of the head and eyes so that they shall act in harmony. Without further discussion it may be stated that whilst minor centres and groups of centres in the cord or elsewhere have certain co-ordinating powers of an order adapted to certain needs and uses, the higher and more complex adjustments are not secured by individual lower motor centres, but these latter act in obedience to higher co-ordinating centres variously situated, but chiefly, though not exclusively, in the cerebellum. In seeking the nervous mechanism of nystagmus, therefore, we are at once directed not to the vestibular nucleus of reception or any single motor nucleus in the medulla or elsewhere, but to the cerebellum. The work of Ferrier,[8] too much neglected by those who have made studies of the semicircular canals, throws much light upon this phase of the subject. Ferrier studied the deviations of the eyes in the monkey and found as follows:

1. Electrical stimulation of the anterior portion of the middle lobe of the cerebellum caused the animal to throw the head backward. This was accompanied by an upward deviation of the eyeballs toward the vertex.
2. Electrical stimulation of the posterior portion or declivity of the upper vermis caused the head to be displaced forward and downward toward the chest, which was associated with a deviation of the eyes downward. Stimulation of the cerebellum caused contraction of the pupils, especially marked upon the side of stimulation.
3. Irritation of the posterior superior lobes upon the left of the median line caused displacement of the head upward and to the left, both eyes turning upward and to the left.
4. Irritation of the posterior superior lobes upon the right of the median line caused upward displacement of the head toward the right, with a corresponding displacement of the eyes upward and to the right.
5. Irritation to the left or right of the pyramid of the middle lobe (pyramis vermis) caused both eyes to deviate to the left or right respectively.
6. Irritation of the posterior extremity of the upper vermiform process (declive monticuli) caused both eyes to deviate straight downward with the electrodes upon the middle of this process. With the electrodes to the left or right of this process the eyes deviated downward and to the left and right respectively.

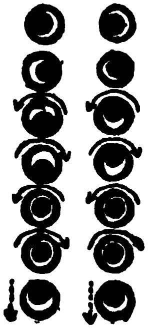
DIAGRAMS ILLUSTRATING REACTIONS OBTAINED BY FERRIER UPON STIMULATION OF VARIOUS PARTS OF THE CEREBELLUM.

IN THE MONKEY



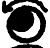
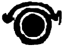
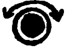
1. Anterior portion of the middle lobe.
2. Posterior portion or declivity of the upper vermis.
3. Posterior superior lobes to left of midline.
4. Posterior superior lobes to right of midline.
5. Pyramid of middle lobe (pyramis vermis) to left of midline.
6. Pyramid of middle lobe (pyramis vermis) to right of midline.
7. Posterior extremity of upper vermiform process (declive monticuli).
8. Posterior extremity of upper vermiform process to left of process.
9. Posterior extremity of upper vermiform process to right of process.
10. Upper vermiform process at its anterior extremity in midline.
11. Upper vermiform process at its anterior extremity to left of midline.
12. Upper vermiform process at its anterior extremity to right of midline.
13. Lateral lobe (semilunar lobule) on the left.
14. Lateral lobe on the right.
15. Flocculus.

IN RABBITS



1. Upper and back portion of middle lobe.
2. Middle and lower portion of middle lobe.
3. Left lateral lobe, upper lobule.
4. Left lateral lobe, middle lobule.
5. Left lateral lobe, lower lobule.
6. Left lateral lobe, anterior lobule.
7. Anterior portion of cerebellum.

IN DOGS

	R.E.	L.E.
1. Pyramid, to the left of it.		
2. Pyramid, to the right of it.		
3. Upper vermiform process, posterior end of declive in midline.		
4. Upper vermiform process, posterior end of declive on left of midline.		
5. Upper vermiform process, posterior end of declive on right of midline.		
6. Lateral lobe, posterior superior lobe on right side.		
7. Flocculus region.		

IN CATS

1. Median lobe, right curve.		
2. Median lobe, left curve.		
3. Upper vermiform process, posterior end of declive in midline.		
4. Upper vermiform process, posterior end of declive to left of midline.		
5. Upper vermiform process, posterior end of declive to right of midline.		
6. Lateral lobe, posterior superior lobule on the left.		
7. Lateral lobe, posterior superior lobule on the right.		

Section of the right vestibular nerve in the sheep (Biehl).		

7. Irritation of the upper vermiform process at its anterior extremity (monticulus cerebelli) (a) in the middle line caused both eyes to deviate directly upward; (b) on the left by the median line caused both eyes to deviate diagonally upward and to the left without rotation; (c) to the right of the median line caused both eyes to deviate upward and to the right.

8. Irritation of the lateral lobe (semilunar lobule) (a) upon the left caused both eyes to deviate upward and to rotate to the left (with the watch); (b) upon the right caused both eyes to deviate upward and to rotate toward the right (against the watch).

9. Irritation of the flocculus caused both eyes to rotate upon their antero-posterior axes.

Experimenting with rabbits, Ferrier found:

1. Irritation of the upper and back portion of the middle lobe of the cerebellum caused horizontal deviation of the eyes to the right, whilst irritation of the middle and lower part caused deviation of the eyes to the left.

2. In the left lateral lobe irritation (a) of the upper lobule caused deviation of the left eye upward, with inward rotation, i.e., against the watch, and downward deviation of the right eye with outward rotation (i.e., against the watch); (b) of the middle lobule caused upward deviation of the left eye with outward rotation (with the watch), and downward deviation of the right eye with inward rotation (with the watch); (c) of the lower lobule caused both eyes to rotate to the right (i.e., against the watch) upon the antero-posterior axes; (d) of the anterior lobule caused rotation of both eyes upon the antero-posterior axes toward the left (i.e., with the watch).

3. Irritation of the anterior part of the cerebellum caused both eyes to deviate upward, followed by vertical nystagmus.

In these experiments Ferrier also noted protrusion of the eyeballs, increased convexity of the cornea, dilatation of the nostrils, movements of the limbs upon the side of irritation, and twitching of the ears.

In dogs Ferrier found:

1. Irritation to the left or right of the pyramid caused deviation of the eyes to the left and right respectively.

2. Irritation of the upper vermiform process, posterior

extremity of the declive: (a) in the middle line caused both eyes to deviate downward; (b) on the left of the middle line caused both eyes to deviate downward and to the left; (c) on the right of the median line caused both eyes to deviate downward and to the right.

3. Irritation of the lateral lobe, posterior superior lobe, on the right side caused both eyes to deviate upward and to the right, with rotation upon their antero-posterior axes to the right (against the watch).

4. Irritation of the flocculus region caused rotation of both eyes on the antero-posterior axes, sometimes to the right and sometimes to the left (i.e., against and with the watch).

In cats Ferrier found:

1. Irritation of the median lobe: (a) right curve, caused horizontal deviation to the right; (b) left curve, caused horizontal deviation to the left.

2. Irritation of the upper vermiform process—posterior extremity of the declive: (a) in the middle caused both eyes to deviate downward; (b) to the left caused both eyes to deviate downward and to the left; (e) to the right caused deviation of both eyes downward and to the right.

3. Irritation of the lateral lobe—postero-superior lobule at various points: (a) on the left caused deviation of both eyes upward and to the left; (b) on the right caused deviation of both eyes upward and to the right.

In addition to the foregoing, irritation of the left side of the cerebellum caused the left pupil to contract, and threw the left limbs into action.

In pigeons Ferrier found no especial movements of the eyes upon irritation of the cerebellum, but irritation of the right or left side of the cerebellum caused the head to be jerked back and toward the side stimulated, and frequently the leg of the same side was brought up and the wing flapped.

With such an array of facts from so reliable an observer, the co-ordinating power attributed to the sixth nucleus lapses into insignificance, and it becomes at once apparent that the connections between the semicircular canals on the one hand, and the motor nuclei of the ocular and other muscles are mainly indirect and effected by paths that traverse the various portions of the cerebellum.

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And since each half of the cerebellum is mainly related to the musculature of the homolateral side, whilst the semicircular canals, as has been already shown, are related chiefly to the musculature of the contralateral side, it follows that the connection between the semicircular canals and the cerebellum is functionally in the main a crossed one. That it is mainly so anatomically, may be gathered from a glance at the relations of the various labyrinthine and other fibre paths roughly indicated in previous chapters. The labyrinthine balanced mechanisms which have been shown to exist, act chiefly through the medium of the cerebellum and the nystagmus, and disturbances of equilibrium consequent upon rotations, aural irrigations and galvanism are produced by enhancement or inhibition (depression) of one or other of the elements of these balanced mechanisms. Thus horizontal nystagmus to the right in cold aural irrigation of the left ear is caused by depression or increased threshold value in the ampullary receptors of the left horizontal canal which causes a relative enhancement or lowered threshold value in the antagonistic ampullary receptors of the right horizontal canal, whereby the normal afferent tonus impulses of the latter acquire an abnormal relative value which is tantamount to stimulation of that portion of the cerebellum on the opposite (left) side which receives them. This causes a steady deviation of the eyes to the left in the horizontal plane as in irritation to the left of the pyramid of the middle lobe of the cerebellum by Ferrier. When, however, the eyeballs have reached a certain degree of deviation to the left, the muscles causing the deviation relax, giving way to the action of their antagonists. These latter by a series of rapid clonic contractions pull the eyes in jerks from left to right for a certain distance when the eyes once more begin moving toward the left under tonic well co-ordinated contractions of the governing muscles. During the steady deviation of the eyes to the left, executed by the left external rectus and the right internal rectus acting conjointly, the opponents of these muscles are in a state of inhibition (reciprocal innervation), such as Sherrington[212] has demonstrated for ocular movements originating in the cerebral cortex. In Sherrington's experiments the inhibition was dependent upon lower centres since it could be elicited after removal of the frontal and occipital areas. Sherrington[47] showed that inhibition is "part and parcel" of the reflex action. The site of inhibition in the simple spinal reflex is central, that is, somewhere in the grey matter, probably at the ultimate synapse between the afferent and efferent (motor) neurones. In reactions from irritation of the motor cortex the seat of inhibition lies probably at their confluence upon the motor neurone, i.e., at the ultimate synapse. It is likely, however, that in other fields of action one cortical element inhibits another (Sherrington[63]). In the reflex compensations incidental to equilibration in man, the commencement of the final common path is, as we have seen, located at centres higher than the spinal neurones, e.g., in centres located in the cerebellum, midbrain, or pons. The commencement of the final common paths for the movements of nystagmus are probably located in the cerebellum. Nystagmus has seldom been elicited from the cerebral cortex (Beevor and Horsley[40]). This, however, does not mean that paths do not originate in the cerebral cortex, which, if irritated are capable of causing nystagmus, for there are free communications between the cerebral cortex and the cerebellar centres.

Nystagmus is a highly co-ordinated complex resulting from associated alternating reflexes. Sherrintgon[47] showed that in such alternating reflexes the antecedent reflex "brings about the stimulus for the next reflex," and predisposes the arc of the next reflex to react to the stimulus when it arrives. In other words at the commencement of the final common path of the succeeding reflex the threshold for afferent impulses is lowered during inhibition. In horizontal nystagmus, therefore, during the steady deviation of the eyes to the left, preparation is being made for the succeeding reflex movements whereby the eyeballs are turned rapidly toward the right. Inhibition plays an important part in this preparation, although afferent impulses, especially those generated in the receptors of the labyrinth and of the ocular muscles (and in the retinal receptors in purely ocular nystagmus) are the immediate determining factor in initiating the movements. Inhibition, it should be remembered, does not always reduce the contraction of the inhibited muscle to zero. This lower grade of contraction in the muscles plays an important part in physiological nystagmus, as it does elsewhere in the muscular mechanisms of the body. Inhibition may give place to excitation, owing to a change of intraspinal conditions. Thus, stimulation of a small afferent nerve often excites a reflex movement of alternating direction, i.e., extension of the knee succeeds primary flexion. Here part of the primary movement was inhibition of the quadriceps extensor, and this was converted into extension of the knee, i.e., excitatory action of the quadriceps extensor. A similar conversion occurs in strychnin poisoning, as well as under the influence of cerebral action (Sherrington[62]).

In purely ocular nystagmus, e.g., that by means of which a person sitting or standing still is enabled to fix visually, rapidly passing objects, alternating reflexes are utilized just as in the case of labyrinthine nystagmus, but in purely ocular nystagmus, stimuli originating in the retinal receptors are the active factors in determining the nystagmus. The afferent retinal paths are in relation with the same final common paths utilized by the labyrinthine receptors. Frequently the labyrinthine and retinal arcs act as allied arcs, as where a person standing or sitting still, views rapidly passing objects. Here a quick turn of the head in the direction opposite to that of the passing objects initiates nystagmus similar in every respect and serving the same purpose as that evoked by the retinal receptors. The relation of alliance between the labyrinthine and retinal arcs is further manifested by the deterioration in function which the labyrinthine mechanism of nystagmus undergoes in persons suffering from complete optic atrophy. In several of these patients with good hearing and good powers of balancing, nystagmus could not be evoked by aural irrigations at 115° F., although some disturbance of the equilibrium with deviation in the gait was effected. Failure to evoke nystagmus was all the more remarkable in these cases, as the patients were comparatively young, e.g., from forty to forty-five years.

Every reflex is purposive (Sherrington), and the two sets of reflex movements that occur in nystagmus have evidently a purposive quality. Thus the "short movements" which give direction to the nystagmus are executed by means of clonic contractions of such rapidity that vision is impossible during their progress. On the other hand the slow movements are executed under tonic contractions so co-ordinated that photo stimuli have time to affect the retinal receptors. If time were allowed for the generation of retinal impulses during the rapid movements of nystagmus, clear continuous vision of moving objects would be an impossibility. Further security from such a difficulty is afforded by frequent rapid interruption of the movement of the eyes under the influence of the short jerks. Hence the latter consist of clonic contractions frequently interrupted by slow movements of short duration and in the opposite direction. Thus even in the excursions of the eyes with the rapid elements, there are short intermissions during which vision is possible. These intermissions avoid the confusion and possible disorientation that might ensue from a lapse in the continuity of vision. The associated movements of the head and upper part of the body may be similarly accounted for by the reciprocal innervation that obtains upon a large scale between wide fields of musculature related to opposing labyrinthine receptors. The commencement of the final common paths for the reflexes involved in these movements are situated most probably in the cerebellum or midbrain. It seems that in extensive movements of equilibration reciprocal innervation obtains between each half of the cerebellum, so that the centres in one half are inhibited when those in the opposite half are stimulated, and *vice versa.* Later we shall see that there is reason to believe that centres in the cerebellum and midbrain are similarly related to centres in the diencephalon with regard to certain gross movements of station and progression.

The paths along which impulses controlling the movements of the eyes travel are variously spread. The afferent labyrinthine connections with the cerebellum have been traced more or less successfully, and the relations have been found to be in the main crossed. With the efferent cerebellar paths, anatomists have not been so successful. Many observations point to the posterior longitudinal fasciculus as carrying numerous efferent cerebellar paths. The observations of Duval and Laborde[30] point to the fact that the fibres of the posterior longitudinal fasciculus connect the third with the sixth nucleus of the same side. Bechterew holds that the fibres to the sixth nucleus are the continuations of those from the cerebellum to the superior olivary nucleus, and Schäfer[11] denies that any fibres pass from the sixth or third nucleus to the posterior longitudinal fasciculus. The conjugate deviation of the eyes that occurs on stimulation of the sixth nucleus is to be accounted for by stimulation of paths for associated movements, and is not to be regarded as an indication of the importance of this centre over other ocular centres in the production of nystagmus. There are numerous other forms of ocular deviations the most rational explanation of which seems to be that based upon the vestibulo-cerebellar relations described in these chapters.

The afferent paths of the vestibular apparatus, in so far as they relate to the complex muscular actions concerned in equilibration with the associated nystagmus and other adjustments, are mainly related directly or indirectly to the cerebellum. It has been seen that vestibular fibres or their continuations reach other structures. Thus the vestibular apparatus is brought into relation with the optic thalamus, the corpora quadrigemina and perhaps the auditory cortex, as well as with the nuclei of other nerves of higher and lower levels. Such connections may supply direct paths for certain co-ordinations, yet these latter are of minor importance compared with those requisite for the maintenance of equilibrium which are carried on mainly under cerebellar influence. The connections of the labyrinth (vestibular nerve) with the cerebellum are established as follows:

1. By paths, interrupted in the vestibular nuclei (including Deiters'), to the roof nucleus (nucleus fastigii) mainly of the opposite side.

2. By way of the vestibulo-olivo-cerebellar tract, i.e., after interruption in Deiters' nucleus (or perhaps some fibres pass without interruption) to the inferior olive of the same side across to the olive of the opposite side, and thence through the restiform body to the cerebellum.

These vestibular relations with the cerebellum are mainly crossed. Thus far we have traced the afferent labyrinthine impulses that cause nystagmus and disorders of equilibrium in rotations, etc., to the opposite half of the cerebellum, stimulation of the different areas of which, in the region of the central lobe, causes phenomena identical with those that are produced by rotations, aural irrigations, and galvanism. The efferent paths along which cerebellar impulses travel to impress the various motor neurones are so scattered that anatomists have not been so successful in tracing them as they have been in the case of the afferent paths. This, however, in no way gainsays the existence of such connections. These efferent paths are therefore grouped with the undefined paths which include: (a) fibres from the cerebellum to the posterior longitudinal fasciculus and thence to other centres and especially to the ocular nuclei; (b) fibres to the cerebral cortex as well as to the various centres in the midbrain, pons, medulla and cord which insure co-ordinated action of ocular, skeletal, and perhaps visceral muscles, as well as of the great centres in the medulla, viz., vagus, vaso-motor, respiratory, etc.

In a general way the efferent cerebellar impulses are distributed to the motor centres situated upon the same side of the body. In the case of eye movements, as in horizontal nystagmus to the left, the slow steady movement is caused by stimulation (relative or absolute) of the area situated on the left of the pyramid of the middle lobe whence impulses are sent by way of the posterior longitudinal fasciculus or other paths to the sixth nucleus of the same side and simultaneously to the group of cells in the third nucleus that controls the internal rectus of the opposite side. Coincident with this stimulation of the area on the left of the pyramid of the middle lobe there is active inhibition of the mechanisms controlling horizontal eye-movements toward the right. The site of this inhibition is at or near the commencement of the final common path, most probably situated in this instance in the corresponding cerebellar centres to the right of the pyramid of the middle lobe. The impulses causing the inhibition pass by collaterals or main stems from the afferent cerebellar (vestibular, etc.) paths that join the corresponding cerebellar centres upon the left side. The chief reason for thus setting up the cerebellar ocular centres of one side in reciprocal relation with the correspond-that the fibres of the posterior longitudinal fasciculus connect the third with the sixth nucleus of the same side. Bechterew holds that the fibres to the sixth nucleus are the continuations of those from the cerebellum to the superior olivary nucleus, and Schäfer[11] denies that any fibres pass from the sixth or third nucleus to the posterior longitudinal fasciculus. The conjugate deviation of the eyes that occurs on stimulation of the sixth nucleus is to be accounted for by stimulation of paths for associated movements, and is not to be regarded as an indication of the importance of this centre over other ocular centres in the production of nystagmus. There are numerous other forms of ocular deviations the most rational explanation of which seems to be that based upon the vestibulo-cerebellar relations described in these chapters.

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As previously stated, the reflex arcs on either side of the body, controlling the movements of horizontal nystagmus, function in an alternating manner so that during the period of inhibition the threshold value is lowered at the commencement of the final common path on the inhibited side. After a time the lowered threshold value, aided probably by summation of subminimal afferent labyrinthine, retinal, or other stimuli, results in conversion of the inhibition into excitation with a corresponding conversion of excitation into inhibition at the commencement of the corresponding final common path on the opposite side. Thus nystagmus has its foundation in alternating reflexes which, when once initiated, tend to continue as each reflex prepares the way for its successor. Alternating reflexes are, however, cut short with ease, hence voluntary fixation of the eyes tends to check nystagmus. This is especially the case where the eyes are strongly deviated to the side toward which the slow elements are directed. The reason why turning the eyes voluntarily to this side is so effective in checking nystagmus rests upon the fact that voluntary fixation is more readily affected in this position owing to the increased cerebellar tonus upon that side which acts as adjuvant to the impulses from the motor cortex of the opposite side. An additional reason is found also, perhaps, in cerebral influence which, like strychnin poisoning, can convert inhibition into excitation in the spinal motor neurones (Sherrington[63]).

In vertical nystagmus toward the feet the anterior portion of the middle lobe is stimulated (relatively or absolutely), with the result that the eyes are drawn steadily toward the vertex. When this excursion has been completed, the alternating return movement toward the feet is executed in jerks, owing to influences, similar to those described for horizontal nystagmus, affecting the posterior portion of the middle lobe in which are located the centres for drawing the eyes toward the feet. It is thus seen that rotations in the mesial plane "face first" and hot bilateral aural irrigations have the same effect as stimulation of the anterior portion of the middle lobe of the cerebellum, whilst rotations in the mesial plane "occiput first" and cold bilateral aural irrigations have the same effect as stimulation of the posterior part of the middle lobe. In vertical nystagmus, the efferent cerebellar impulses reach the nuclear group on either side of the middle line controlling the muscles involved in the movements.

In rotary nystagmus the cerebellar areas affected are located in the region of the flocculus and lateral lobe (semilunar lobule). In rotary nystagmus against the watch the efferent impulses controlling the slow element are sent to the fourth nucleus on the left side which, on account of the decussation of the fourth nerve, supplies the right superior oblique. At the same time corresponding impulses are sent to the group of cells in the third nucleus controlling the left inferior oblique. When the excursion in the direction of the hands of the watch has been completed under the influence of stimulation of some area in the region of the flocculus or lateral lobe (semilunar lobule), the return movement in the direction against the hands of the watch is made in rapid jerks owing to influences, similar to those described for horizontal nystagmus, affecting analogous areas in the flocculus or lateral lobe upon the opposite side which control the muscles involved in the movement, i.e., the left superior oblique and the right inferior oblique.

It may be noted that the direction of the movements of the eyes in rotary nystagmus is, at times, difficult to determine, for frequently the jerky motions are slow enough to permit actual vision and strongly turning the eyes in one direction or another may so alter the movement that study with the ophthalmoscope becomes difficult or fruitless.

The efferent cerebellar paths involved in vertical and rotary nystagmus probably pass by way of the superior peduncle and posterior longitudinal fasciculus. It is not necessary, as Schäfer seems to think, that the fibre paths of the posterior longitudinal fasciculus be traced outward from the motor nucleus toward the muscle. It is sufficient if the fibres reach the nucleus and impress the motor neurones.

The flexible mobility of the eye is such that deviations and rotations can take place to a limited extent in almost every conceivable direction. It is, therefore, highly probable that whilst, in the main, the ampullary receptors of each semicircular canal are chiefly affected by rotations in one plane, they are also to a lesser extent perhaps affected by movements in several other planes, each of which in turn is associated with definite cerebellar centres for ocular tonus control. The grounds for this assumption are to be found in the diagonal deviations in Ferrier's experiments on stimulating certain cerebellar areas, e.g., to the left of the middle line in posterior superior lobes, and in the diagonal nystagmus noted upon rotations in diagonal planes, traces of which are occasionally met with in aural irrigations and in galvanism.

The cerebellar balanced mechanisms are so related to the cerebral mechanisms that each half of the cerebellum acts in conjunction with the opposite cerebral hemisphere. Thus each half of the cerebellum controls the muscles that turn the eyes in a certain direction, e.g., in lateral movements toward the same side and inhibits the muscles that move the eyes in the opposite direction. The centres for certain movements (e.g., the vertical) are, however, not represented in one side of the cerebellum as opposed to the other, but are grouped about the median line in one portion of the vermis, whilst the centres for the antagonistic movements are grouped about the middle line in another area of the vermis. The cerebellar mechanisms act purely in a reflex manner. Their afferent and efferent limbs have widespread connections on account of the extensive influence the cerebellum exerts on various muscular activities.

The chief afferent cerebellar paths are those related to the labyrinthine and retinal receptors, and to the cerebral cortex, and the peripheral structures concerned in the muscle sense (Golgi-Mazzini organs, etc.). The relations of the cerebellum to the labyrinth, organs of muscular sense, and to the various nuclei of the sensory nerves have been sufficiently discussed. The relations to the cerebral cortex are made through paths that convey impulses in both directions. Stimulation of the frontal ocular area sends impulses to the opposite half of the cerebellum, after the manner in which labyrinthine impulses reach the contralateral half of the cerebellum. Probably afferent cerebellar impulses which originate in various parts of the cerebral cortex, e.g., frontal, temporal, and occipital, normally control the related cerebellar mechanisms. The cerebral cortex in this instance acts somewhat after the manner of sensory peripheral mechanisms in relation to the cerebellum. Risien-Russell's [60] observations seem to warrant this conclusion. This physiologist removed part of the frontal ocular areas and subsequently removed the lateral lobe of the cerebellum on the same side. In this instance the last part of the operation was followed by much less deviation of the eyeballs to the sound side than would be obtained ordinarily by removal of the lateral lobe of the cerebellum alone.

The efferent limbs of the cerebellar mechanisms are scattered widespread on their way to the different spinal motor neurones which they influence. They cannot therefore be readily differentiated anatomically from the afferent limbs. In a general way, however, they are related to the muscles upon that side of the body in which the cerebellar neurones from which they spring are located.

Efferent cerebellar paths also carry impulses to the various regions of the cerebral cortex from which in turn spring afferent cerebellar paths. It is by means of these paths running in both directions between the cerebrum and the cerebellum that these two organs act in harmony in the complex processes involved in ocular movements, and in movements of equilibration. Risien-Russell, [60] as before stated, has shown that the cerebellum exerts a marked influence upon the related centres in the cerebral cortex one-sided lesion of the former causing increased irritability in the opposite cerebral hemisphere. Luciani, however, concluded that excitability was increased in some points and in others depressed. It is by means of this functional relation that the cerebrum is enabled to compensate most economically for temporary or permanent defect of the cerebellar mechanisms. This relationship perhaps accounts, in part, for the well-known defective cerebral development which accompanies congenital cerebellar defects. It also seems to have some bearing upon the nystagmus and vertigo seen at times in patients suffering from manic-depressive psychosis, although in the only case observed by the author there was also present a slight aural defect. In this case which was of the mixed type, the probable explanation of the phenomenon was that compensation for the defect in the vestibulo-cerebellar mechanisms had been made but the supervention of the manic-depressive psychosis acted somewhat after the manner that ether or chloroform narcosis acts in dogs in which compensation has been established after removal of portions of the cerebrum or cerebellum, causing a reappearance of the cerebellar or cerebral defect. No opportunity was afforded of studying thoroughly the labyrinthine reactions.

Besides the nystagmus there were other signs of cerebellar disturbance, such as vomiting, vertigo, and general muscular weakness. The nystagmus disappeared with these in a short time (about two weeks) as the patient improved somewhat in general mental and physical health.

The conclusions are:

1. That nystagmus in rotations is a co-ordinated movement, executed reflexly by means of afferent labyrinthine impressions acting upon centres situated in and about the middle lobe of the cerebellum and intended to aid in the compensatory adjustments of rotations as well as in the visual fixation of rapidly passing objects.

2. That the ampullary receptors of each semicircular canal have definite relations with certain portions of the cerebellum in the vicinity of the middle lobe, viz., the ampullary receptors of the left and right horizontal canals with the centres situated to the right and left respectively of the pyramid of the middle lobe (pyramis vermis); those of the paired posterior and superior canals with the centres situated about the middle line of the posterior and anterior portions respectively of the middle lobe; and those of the superior and posterior canals taken individually or

in crossed pairs with the centres located in the region of the flocculus and lateral lobes.

3. That by means of these relations and the corresponding nystagmus a guide may be had in localizing peripheral vestibular nerve lesions and lesions of the cerebellum.

CHAPTER XXI

RELATIONS OF THE SEMICIRCULAR CANALS TO THE OCULO-MOTOR NUCLEI AND THEIR BEARING UPON THE RESULTS OF CERTAIN EXPERIMENTS

The phenomena following section of the vestibularis in the sheep (Biehl[70]) have already been somewhat discussed. It remains to consider the ocular phenomena in the light of the relations, just established, of the semicircular canals to the oculo-motor nuclei. The crossed relation of each labyrinth to the fourth nucleus and that portion of the third nucleus governing the action of the associated inferior oblique afford a ready explanation for the skew deviation. A glance at the diagram of these relations, page 283, shows that section of the right vestibular nerve cuts off the afferent vestibular tonus impulses to the VI and IV nuclei on the left side, as well as those to that part of the III nucleus controlling the right internal rectus and the left inferior oblique. The effect is:

1. Paresis of the left external rectus and of the associated right internal rectus, causing a general tendency of the eyeballs to turn toward the right.

2. Paresis of the right superior and left inferior oblique, thereby leaving the eyeballs under the unopposed control of the right inferior and of the left superior oblique in conjunction with the right rectus externus and the left rectus internus.

The net result is that the right eye is drawn downward and outward, and the left eye upward and inward. (See diagram.) In section of the vestibular nerve in the dog, Bechterew[69] observed similar deviations of the eyes. Section of both vestibular nerves causes no ocular deviations —the eyes assuming the primary position.

The recorded phenomena following irritation of the vestibular nerve are, for the most part, untrustworthy.

The twofold element of motion in the nystagmus that accompanies irritation of the vestibular nerve has been a source of confusion, so that many observers reached widely divergent conclusions. Hence the phenomena attributed to irritation of the canals or of the divided vestibular nerve have to be taken with extreme caution. In the study of this phase of the question it seems that clinical methods have the advantage and are more trustworthy than those of the laboratory. This should be an omen and incentive to the mere practitioner.

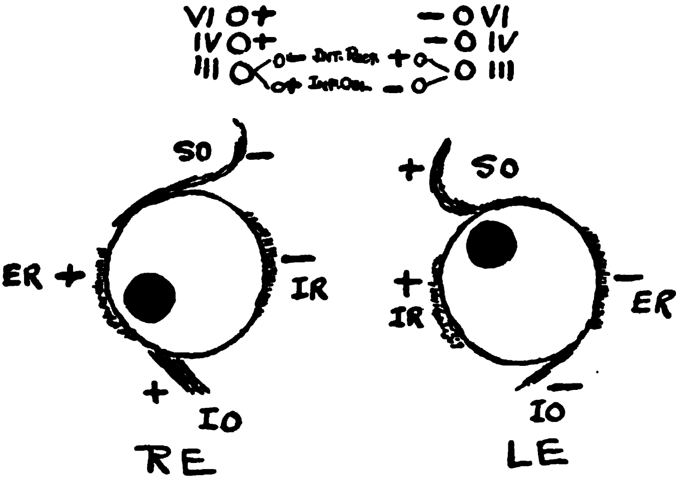


Diagram showing the effect upon the ocular muscles in section of the right vestibular nerve. The minus sign indicates absence of vestibulo-cerebellar, i.e., labyrinthine tonus, and the positive sign indicates a relative excess of vestibulo-cerebellar tonus. III, IV, and VI indicate the motor nuclei of the eyeballs. SO, superior oblique; IO, inferior oblique; IR, internal rectus; ER, external rectus; RE, right eye; LE, left eye.

Sheep and dogs, in which the vestibular nerve upon one side has been divided, endeavour to lie upon the side of operation, for in this position the actual tonus effect of the opposite labyrinth is reduced to a minimum, the muscles under its control being at rest. There is therefore less need for tonus innervation from the labyrinth on the side of operation. In this position the limbs of the side of operation were relaxed, whilst those of the opposite side were lengthened (and stiff) (Bechterew [69]) due no doubt to cerebral motor influence, as Sherrington [63] observed that lying upon the side tends to relax (the reflex) tonus of the extensor muscles. Lying on the back the difference between the two sides was less apparent. In positions upon the side, therefore, there is evidence of some functional activity of the labyrinthine mechanisms probably concerned in slight equilibratory adjustments. This point is emphasized in seasickness and related conditions, where turning from the back upon the side may be sufficient to determine impending vomiting. When the animal lying on the side of operation lifted its head from the ground or made the slightest movement, nystagmus occurred or if already present it became aggravated. If the animal recovered sufficiently to stand it was easily pushed over from the sound side, whilst the immediate effects of the operation were rolling movements about the long axis to the side of operation. Hence there was, after section of the right vestibular nerve in the sheep, a weakness or deviation of the head and body toward the side of operation, with a tendency to rotate in the same direction (in man, from right to left and with a mixed nystagmus the horizontal element being directed to the left and the rotary element with the watch, all of which correspond exactly with the phenomena of cold irrigation of the right ear, or of hot irrigation of the left ear, or with the primary phenomena of rotations about the long axis from right to left or the secondary phenomena of similar rotations from left to right; or with the phenomena that accompany closure of the circuit with the anode over the right mastoid or with the cathode over the left mastoid. The muscles which execute these movements are mainly situated upon the right side (i.e., on the side of operation) and are mainly in relation with the labyrinth of the opposite side. Normally, impulses from each labyrinth counterbalance each other in the adjustments necessary for the maintenance of equilibrium. Any material interference with the labyrinthine receptors, whereby the threshold value of stimuli originating normally in response to movements or displacements of equilibrium are enhanced or depressed on one side or the other will cause disturbances similar to those following section of the vestibular nerve.

The constant tonus effect of the labyrinth upon the muscles has therefore been demonstrated. The crossed functional relation between the labyrinth and the musculature of the opposite side must be admitted (though v. Bechterew denies this), and finally, the evidence seems overwhelming that the main functional and anatomical relation between the labyrinth and the cerebellum is a crossed one.

This view of the crossed relation of the vestibular apparatus receives corroboration from the results of various observers. There is anatomical and physiological evidence that the cerebellum is in crossed relation with the cerebral cortex, optic thalamus, red nucleus and other important centres, and with the peripheral organs for afferent visual, labyrinthine, including auditory, and perhaps to some extent, tactual, muscular and other impressions.

Luciani[61] and Risien-Russell[60] observed phenomena similar in general to those that follow section of the vestibularis, but with the movements toward the sound side after ablation of one-half of the cerebellum. Section of the middle cerebellar peduncle causes rolling movements toward the sound side (v. Bechterew, Schiff, Lussana, Longet and others). Here it should be noted that the afferent middle peduncular paths, i.e., to the cerebellum, are severed mainly after decussation, whilst the efferent paths of the peduncle which, through double decussation, are mainly in relation with the muscles on the same side of the body are cut before any decussation has taken place. The resulting movements are the same as those which would follow section of the same afferent paths on the other side of the body before decussation had taken place. In this latter event the movements would be toward the side of injury as in the case of section of the vestibularis. Again, section of the vestibular paths, after crossing the middle line, if such a thing were feasible, would beget movements toward the sound side which is exactly what follows ablation of one-half of the cerebellum. After section of the vestibularis, stimulation of the proximal stump causes rotation of the body on the long axis toward the sound side. This points to a relatively enhanced cerebellar tonus upon the side related to the stimulated stump and consequent determination of the body movements toward that side, i.e., toward the sound side.

Section of the superior cerebellar peduncles causes no distinct disturbances of equilibrium (v. Bechterew[13]). However, circular, instead of rolling movements were observed with lateral deviation of the eyes toward the side of operation after section between the cerebellum and corpora quadrigemina, whilst section between the corpora quadrigemina and the optic thalamus caused deviation to the healthy side with nystagmus. Section of the fibres passing below the Sylvian aqueduct in their course from the postero-lateral wall of the third ventricle to the antero-external part of the floor of the fourth ventricle, as well as section of the middle cerebellar peduncle caused rolling movements in the direction of the sound side (v. Bechterew [13]). Ferrier[8] believes these phenomena were caused in reality by interruption of the paths in the superior peduncle or by injury of the oculo-motor nuclei or by a combination of both of these factors. The phenomena observed by v. Bechterew in experimental lesions of the third ventricle were such as led him to believe that in this ventricle resides another organ of equilibrium. As the third ventricle is so closely related to the optic thalami the relations of which are in turn so widespread, and to afferent optic paths on their way to the cerebellum (Panse[218]), it is very probable the phenomena were due to lesions of the optic fibre paths or of paths to or from the optic thalami. Moreover the fibres and nucleus of the posterior longitudinal fasciculus invade to some extent the territory of the third ventricle.

As the superior peduncles begin to decussate at the level of the lower (caudal) border of the inferior colliculi, it is evident that in v. Bechterew's division of the peduncle between the corpora quadrigemina and the cerebellum, and between the corpora quadrigemina and the optic thalamus the effect of the lower section on one side would be practically equivalent to section at a level above the quadrigemina on the other side. v. Bechterew believes the superior cerebellar peduncle completely decussates. The most generally accepted opinion is that most of the fibres of the peduncle cross at the decussation. Above the decussation most of the fibre paths are interrupted in the red nucleus, those not so interrupted passing through the subthalamic region to end about the cells of the thalamus. Most of the fibre paths interrupted in the red nucleus are continued by rubral neurones to the thalamus. From the thalamus the uninterrupted paths, as well as those interrupted in the red nucleus, are continued to the cerebral cortex. Many of the fibre paths interrupted in the red nucleus, however, are diverted into the rubro-spinal tract, which traverses the brain stem and antero-lateral ground bundles of the cord to reach the ventral root cells. These fibres, owing to Forel's decussation, recross the middle line thus bringing the half of the cerebellum in which the fibre paths originate into relation with the muscles of the same side of the body. It does not matter at what point the reflex cerebellar tonus arc is broken, whether in the afferent or efferent limb the effect is the same, i.e., loss of tonus of the muscles supplied, and consequent disturbance of functional precision in other co-ordinating centres, especially those situated in the optic thalamus and cerebral cortex. Hence section of the superior peduncle by Ferrier, and lesions of the optic lobes, as well as of the fibres connecting them with the oculo-motor nuclei were followed by movements toward the healthy side. Since the lesion in section of the superior peduncle is situated at a point where decussation has already taken place in the case of the afferent paths, and where it has not yet occurred in the case of the efferent paths, which, in the case of paths making for the spinal cord owing to a double decussation, are in relation with the muscles of the same side of the body, the phenomena are altogether analogous to those following hemi-extirpation of the cerebellum of the same side or section of the middle peduncle of the same side or section of the vestibular nerve after decussation, if such a thing were possible. Interference with the fibre paths of the superior peduncle destined for the thalamus and cerebral cortex causes disturbance of the cerebello-cortical circuit, whereby the animal is misinformed not only as to his relations in space, but also as to the degree of innervation necessary to control certain voluntary movements in the adjustments of equilibration. This tends to aggravate the vertigo caused from interference with the other parts mentioned above (afferent and rubro-spinal paths). The result is that the animal in a general way moves or rolls in the direction of the sound side, the musculature of which is in relation with the intact functioning side of the cerebellum. This brings that particular side of the cerebellum to a condition of rest when the sense of vertigo is at a minimum. Again this is exactly what the sheep or dog with the divided vestibular nerve does, viz., seeks that position in which the muscles connected with the half of the cerebellum that has all the afferent and efferent paths of its tonus mechanisms intact, thereby reducing to a minimum the subjective and objective disturbances that spring from injury to one or other of its main afferent or efferent paths. The site of the lesion, i.e., whether it is situated on one side or the other of the median line is not what determines the direction of the movement, for in many instances the same fibre paths may be divided before or after crossing the middle line. The cerebellar centres involved are the all-important thing in determining the direction of the ocular and bodily deviations which, in a general way, are always away from the side of the cerebellum with which the injured afferent or efferent paths are connected. If this rule be kept in mind, the explanation of clinical and experimental phenomena will be greatly facilitated. The conjugate deviation seen in extensive cerebral hemorrhage can be readily explained when the intimate relation of each half of the cerebellum with the opposite cerebral hemisphere is understood. A large hemorrhage over one cerebral hemisphere not only inhibits the motor centres in the cerebral ocular areas on that side, but cuts off all or many of the impulses to and from the cerebellum by way of the related superior and middle peduncles. The result is equivalent to section of such peduncles after decussation, which means suspension to a certain extent, of the reflex activity of the related cerebellar centres on the sound side. The half of the cerebellum on the side of the cerebral lesion has unrestricted play, and the related ocular mechanisms by their enhanced relative tonus to the oculo-motor muscles tend to turn the eyes in a general way toward the side of the cerebral lesion. The turning of the eyes in this instance toward the side of the lesion is analogous, in general direction, to the turning of the eyes that follows section of the vestibular nerve, that is, toward the side of the lesion, but away from the side of the cerebellum in which the centres affected by the lesion are located. The turning of the head in the conjugate deviation of cerebral apoplexy is due to suspension of function of centres located near the frontal ocular centres with which they frequently act in conjunction, as in the common associated movements of turning the head and eyes to one side or the other. Ferrier[8] found that stimulation of the general frontal ocular area on one side caused the head and eyes to turn toward the opposite side. Later, Beevor and Horsley[40] separated the centres for the head movement from those of the eye movement. Destruction, therefore, of this region or inhibition of the centres contained therein, naturally causes a turning of the head and eyes toward the side of the lesion. It should be stated, however, that Longet,[271] Lafarge,[272] Lusanna,[273] and others found that section of the superior peduncle, after it has crossed the middle line as it lies in the tegmentum, caused rolling on the long axis to the sound side, whilst section of the peduncle before decussation near its exit from the cerebellum caused rotation to the side of the lesion. In estimating the import of these and all other operations upon the cerebellum or its peduncles, the distinction between phenomena attributable to irritation and those attributable to destruction must ever be kept in mind as well as the distinction between movements which are purely automatic and those due to voluntary compensation.

We have seen that the experimental phenomena connected with lesions of the cerebellum and of the superior and middle peduncles, as well as with section of the vestibular nerve, point strongly to the fact that the peduncles mentioned contain paths for the transmission of impulses concerned in the reflex acts of equilibration that are associated with the cerebellar centres. These paths are similar to those already found in the vestibular nerve but the peduncles contain efferent as well as afferent paths. The phenomena observed also point to a crossed cerebellar relation in these as in the labyrinthine paths and anatomical investigations lend corroboration. When we approach the study of the inferior peduncle, difficulties are encountered which spring from the complexity of its structure coupled with the inevitable lack of uniformity in the experimental findings.

v. Bechterew[18] found that section of the inferior peduncle in any part of its course from the olive to its entrance into the cerebellum, as well as injury to the olive on the same side always produced rolling movements toward the side of operation. This is one of v. Bechterew's chief reasons for maintaining that the vestibular nerve is mainly in relation with the homolateral half of the cerebellum. The extreme complexity of the fibre paths in the inferior peduncle render it difficult to deduce reliable conclusions from lesion of this structure. The proximity of Deiters' nucleus and of the vestibular paths to and from it, including vestibulo-olivary paths from Deiters' nucleus to the olive, not only explain the phenomena, but turn them to account as strengthening the assumption that the olives are, in part at least, relay stations for afferent vestibular impulses (vestibulo-olivo-cerebellar) as well perhaps as for other cerebellar (afferent and efferent) impulses.

It has been assumed that Helweg's fasciculus is related to the inferior olive. Such a relationship affords opportunity for its decussation and the relaying of afferent cerebellar paths.

Many physiologists, however (Rolando,[274] Magendie,[228] and others), have found fairly constant and uniform results after section of the inferior peduncle. One of these results was a tendency to roll to the side of the lesion.

Notwithstanding the diversity of opinions as to the direction in which the animals tend to roll after section of the various peduncles, or after removal of one-half of the cerebellum, the deviation of the eyes is always directed to the side on which the cerebellar centres and their related afferent and efferent paths are intact. This fact serves as a guide and helps us to avoid those errors which must inevitably spring from confounding phenomena due to irritation with those due to destruction and automatic with voluntary compensatory movements.

CHAPTER XXII

ON OCULAR MOVEMENTS AND NYSTAGMUS

Ferrier and Munk, working independently of each other, first located the centres for ocular movements in the frontal region of the cerebral hemispheres, viz., in the posterior half or two-thirds of the superior and middle frontal convolutions. Stimulation of this area causes movements of the head and eyes toward the opposite side.

Horsley and Schäfer[219] confirmed Ferrier's results and got movements from a more extended area. Beevor and Horsley[40] separated the area for movements of the head from that for movements of the eyes and further differentiated the lateral movements of the eyes. They found that turning both eyes in the horizontal plane toward the opposite side was rare as a primary movement and best represented in the convolutions in front of the paracentral sulcus and to a less extent behind it. Movement of both eyes to the opposite side and upward was found to be rare as also movements of both eyes to the opposite side and downward. Limited rotation of the eyes sufficient to restore the direct position of the visual axes was frequently observed. Nystagmus was occasionally observed on stimulation of areas in front of the precentral sulcus. Schäfer[15] discovered in the occipital region centres for ocular movement similar to those found by Ferrier in the frontal region. Ferrier[8] had, however, already obtained movements of the eyes from irritation of the occipital areas, but considered them as merely associated movements. Sherrington[212] after division of the third and fourth cranial nerves upon the left side, observed conjugate deviation of both eyes to the right upon excitation of the cortex on the left side, viz., Ferrier's ocular centre in the frontal region and Schäfer's centre in the occipital region. Both eyes turned to the right, but the left only went as far as the middle line.

Sherrington also divided the sixth nerve on the left side and found that stimulation of the frontal cortex of the right hemisphere caused the right eye to move to the left sharply, whilst the left eye did not move, or moved sluggishly up to the full primary position. Sherrington concluded that the straight muscles of the eye can be inhibited by appropriate excitement of certain parts of the frontal, and still better of the occipital cortex on the side opposite to that of the muscle. He found that this inhibition takes place on stimulation of the corona radiata and occipital radiations after removal of the frontal and occipital cortex. He further noted that on severance of the third, fourth, and sixth nerves, the eyeball assumed the primary position.

Schäfer and Mott[220] demonstrated that movements other than lateral are represented in the cerebral cortex. Risien-Russell,[60] working independently, also demonstrated the same fact in an interesting series of experiments undertaken especially for the investigation of ocular movements.

In monkeys, after division of the internal rectus on the side of cortical stimulation, and of the external rectus upon the opposite side, he observed up and down movements of the eyeballs on irritation of the frontal ocular area. By practising this method of severance of certain muscles the following movements were shown to be related to certain areas in the frontal (and presumably also in the occipital) cortex:

1. Direct movement of both eyes downward; centre just above horizontal fissure of the central sulcus.

2. Direct movement of both eyes upward; centre slightly above and in front of the preceding area.

3. Movement of both eyes downward and to the opposite side; centre in front of vertical part of the precentral sulcus just below the line of the posterior extremity of the horizontal sulcus.

4. Movement of both eyes upward and to the opposite side; centre just below the horizontal fissure of the precentral sulcus.

5. Movement of convergence; centre not constant, but generally in front of centre described under No. 3.

6. Direct lateral movement to the side of stimulation (exceptional).

7. Movement upward and to the same side (exceptional).

Russell next proceeded to practise removal of the various areas in the frontal cortex corresponding to the location of the centres for the various eye movements. The operations caused conjugate deviation of the eyes to the side of the lesion. In time the deviation was corrected, but returned when the animal was anæsthetized just before the stage of surgical anæsthesia, whilst the corneal reflex was still present. With the advent of profound narcosis, each eyeball moved to a position of slight outward deviation which is the normal position for dogs in deep narcosis. A similar divergence of the eyes with independent motions was observed by Mercier and Warner[221] in man in deep coma from anæsthetics or other cause. In recovery from anæsthesia, the animal's eyes went into conjugate deviation toward the side of the lesion and remained thus for one to two hours after restoration to consciousness. The generally accepted view as to the causation of conjugate deviation after removal of the cerebral ocular centres, is that it results from the unopposed action of the structures upon the sound side.

Risien-Russell attempts to explain the recovery from deviation after operation. There are, according to him, four possibilities to account for the phenomena:

1. The remaining parts of the ocular centres take on action.
2. Increased supply of nervous influence from the sound hemisphere.
3. Cerebellar compensation.
4. Diminished output of energy on the sound side to the related muscles.

The first hypothesis is rejected because compensation occurs even where the whole cortical oculo-motor area of one hemisphere is removed. The author does not state whether the removal included the occipital as well as the frontal areas, but adds that ocular movements may be represented in lower areas, e.g., in the leg and arm regions.

The second hypothesis is rejected as unlikely because movements to the side of stimulation are exceptional.

The third hypothesis is not so easily dismissed because of the findings of Ferrier[8] and Luciani.[61] The former found on irritation of certain areas of the cerebellum that the eyes turned to that side, whilst the latter found on ablation of one lateral half of the organ that the eyes turned to the opposite side. These observations show that each half of the cerebellum exerts an influence upon the ocular movements similar to that exerted by the opposite cerebral hemisphere. It is therefore possible that the cerebellum may compensate for loss of the cerebral ocular centres and so correct the conjugate deviation.

The fourth hypothesis seems to be the one toward which Russell most inclines. The amount of motor energy liberated by any centre is proportioned to the resistance to be overcome. After destruction of one area, the muscles related to the corresponding area on the opposite side have less demand put upon them through the reflex centres, and their related centres (on the sound side) gradually accommodate themselves to the new conditions, i.e., become less active.

It should be noted that this is a purely negative sort of recovery at the expense of the function of active ocular movement. Sherrington's observation, that the cerebral cortex not only controls the straight ocular muscles which turn the eyes to the opposite side, but actively inhibits their antagonists, is distinctly opposed to this view. Moreover, the reappearance under anæsthesia of the deviation which had been recovered from, cannot well be explained on this hypothesis. The same objection holds for cerebellar compensation. If the cerebellar mechanisms be included, the most plausible explanation is afforded by hypothesis No. 1. Thus it is known that ocular movements are represented in the frontal and in the occipital areas, and it may well happen that eye movements are represented in other areas.

The compensation for conjugate deviation on this hypothesis therefore takes place by the gradual acquirement of active participation of other areas of the cerebral cortex on the side of the lesion. Under anæsthesia the newly acquired functions are the first to be suspended, thus permitting the reappearance of the conjugate deviation.

When complete anæsthesia supervenes all the ocular centres are in abeyance, and on coming out of the anæsthetic, the centres that were overcome last are the first to recover, causing thereby the reappearance of conjugate deviation. The ocular centres of both cerebral hemispheres do not, in normal dogs, succumb simultaneously in ether or chloroform narcosis (Risien-Russell). This fact does not necessarily contravene the view just expressed which relates more to apes and the higher forms; for in the dog ocular and other movements are but feebly represented in the cerebral cortex, and the movements so represented are probably not equally represented in each hemisphere. Thus in dogs and cats, besides the lateral movements, the only movements represented in the frontal cortex are the upward movements, whilst in monkeys all sorts of movements are represented (Risien-Russell). This clearly shows the relative automaticity that obtains in ocular and other movements in the dog and lower forms as pointed out by Ferrier. It also emphasizes the relative importance of the mesencephalic, cerebellar, and other automatic centres in these lower forms.

It has long been known that the cerebellum influences ocular movements.

Saucerotte[222] (1769) concluded from his experiments that the eyes receive innervation from the cerebellum.

Magendie[223] (1824) described the position of the eyes after lesions of the cerebellum and of the middle peduncles. In rabbits he found on section of one middle peduncle the eye on the same side turned downward and forward whilst that of the opposite side turned upward and backward. (See diagrams.)

This position of the eyes is somewhat similar to the position observed by Biehl[70] on section of the right vestibular nerve in the sheep, the only difference being that in the latter the position of the left eye is raised and that of the right lowered. When the corresponding peduncle of the opposite side was cut, Magendie found that the ocular deviation disappeared and the eyes reverted to the primary position.

Gratiolet and Leven[224] (1860) incised the cerebellum vertically through the centre of one lateral lobe and observed that the eye on the same side turned downward and backward, whilst that on the other side turned upward and forward. Leven and Ollivier[225] passed needles through the skulls of guinea-pigs into the cerebellum, and observed strabismus. Renzi [226] (1864) concluded that in fishes the cerebellum influenced vision by regulation of the co-ordinated voluntary movements of the eyeballs. Weir-Mitchell [62] (1869) observed that most deep lesions of the cerebellum produced strabismus. Ferrier [8] found that irritation of areas of the cerebellum in and about the vermis caused various movements of the eyes, the general direction being toward the side of irritation. He also observed contraction of the pupils, especially on the homolateral side, bulging of the eyeballs, and movements of the limbs, etc., upon the side of irritation. Luciani [61] (1891) found on extirpation of one-half of the cerebellum that the eyes turn to the opposite side. He also observed strabismus and nystagmus after removal of different parts of the cerebellum. Risien-Russell on removal of one lateral lobe of the cerebellum in dogs and cats observed a skew deviation of the eyes, the eye on the side of the lesion turning upward and forward, whilst the opposite eye turned downward and backward. When the effects of the anæsthetic had passed off, the eye on the side of the cerebellar lesion went back to the primary position, whilst the eye on the opposite side maintained its abnormal position for several days. Removal of both lateral lobes, leaving the middle lobe intact, caused both eyeballs to rotate downward and to a variable extent outward. Excision of half the posterior part of the middle lobe caused the eye of the same side to rotate downward and outward. Excision of the whole of the posterior part of the middle lobe caused both eyes to turn downward and slightly outward. Total ablation of the cerebellum caused a variable amount of downward rotation of both eyeballs. All these ocular deviations were recovered from in a variable length of time. After recovery the deviations were re-established during narcosis.

Two forms of nystagmus were observed:

A spontaneous form which was of a definite direction irrespective of voluntary ocular movements; and nystagmoid jerks which occurred only when the globes were moved voluntarily, the direction of the jerks being the same as that in which the globes were moved.

Ablation of one lateral lobe caused nystagmus which was of more or less constant occurrence, and which lasted for a variable time after the operation at times only for a day or two. The nystagmus was always lateral and consisted of slow jerks toward the side of the lesion. The jerks were more marked in the eye on the sound side.

Removal of both lateral lobes caused nystagmus of upward direction with a tendency of the upper segment of each globe to rotate from within outward at first. This spontaneous nystagmus was replaced by nystagmoid movements which occurred only when the eyes were moved. The direction of these nystagmoid movements was that in which the eyes were moved. In the course of two or three days all nystagmus had disappeared.

Extirpation of one lateral half of the posterior part of the middle lobe caused nystagmus of both globes. The jerks were toward the opposite side and slightly upward.

Ablation of the whole posterior part of middle lobe caused vertical nystagmus with occasional irregular rotatory movements.

Ablation of the whole cerebellum did not cause spontaneous nystagmus, but there appeared nystagmoid jerks of both globes in the direction of the voluntary ocular movements. After recovery from the nystagmus following cerebellar lesion, ether or chloroform narcosis caused a reappearance of the nystagmus just as the globes were about to turn to one side or the other. It ceased as the anæsthesia deepened and the abnormal (normal for narcosis) position of the eyes was established. This was seen several months after ablation of one lateral lobe.

By way of control, experiments were made by: 1, extirpating the labyrinth on one side; 2, by intra-cranial section of the eighth nerve; and 3, by chemical irritation of the eighth nerve on one side. All these procedures, according to Risien-Russell, were followed by similar results, viz., rotation of the eye upon the side of the lesion downward. There was doubt as to whether there was also an additional displacement outward as described by v. Bechterew, or inward, sometimes the one and sometimes the other being observed.

The eye on the sound side was turned inward but without the upward rotation described by v. Bechterew.

Nystagmus was always marked just after the operation, the direction being upward with a varying amount of inward element in the eye on the side of the lesion, and lateral, with the jerks outward, in the eye of the sound side. It was noted that two or three hours after the operation there might appear rotary nystagmus of both eyes in which the upper segment of the eye on the side of operation rotated inward, whilst that of the opposite eye rotated outward.

Ablation of the frontal ocular area of one hemisphere, followed later by extirpation of the opposite lateral lobe of the cerebellum. See diagrams (pp. 329, 330). Stages:

1. In narcosis before operation the eyes were turned to the left.
2. Removal of part of ocular area of the right hemisphere caused both eyes to turn to the right.
3. In a few days the eyes had resumed their normal position.
4. Removal of left lateral lobe of cerebellum caused the right eye to turn to the right with a downward inclination. The left eye was unaffected.

Hence removal of one lateral lobe of cerebellum in an animal deprived of part of its frontal ocular area of the opposite cerebral hemisphere has the same result as the same operation done upon an animal with the cerebral ocular areas intact. Ablation of part of the eye area of one hemisphere was followed later by extirpation of the lateral cerebellar lobe of the same side. The effect of the second operation was to cause the eye on the sound side to turn outward slightly, certainly to a much less extent than a similar operation on dogs with intact cerebral hemispheres.

This experiment indicates clearly the functional dependence of the lateral cerebellar lobe upon the related cortical area of the opposite cerebral hemisphere. Assuming that compensation after the first operation had taken place by the active intervention of the other ocular centres in the right hemisphere, it may also be assumed that sufficient time had not elapsed for the new afferent cerebro-cerebellar paths to function properly. The effect of removal of the cerebral ocular area on the right side may properly be compared with the effect of section of the right vestibular nerve, in that it withdraws the afferent cerebellar tonus impulses from the left half of the cerebellum, so that when the antagonistic centres in the right half of the cerebellum are removed, little or no deviation of the eyes occurred. The fact that there was some deviation of the left eye toward the left, seems to prove that this latter was not due to the inequality of direct cortical innervation to the ocular muscles, inasmuch as the deviation due to the first operation had already been compensated. On the other hand it might be said that this compensation was effected through overactivity of the left, or underactivity of the right half of the cerebellum. The reappearance of the ocular deviations in narcosis and other considerations, e.g., the slight extent of the deviation of the left eye after the second operation, render this extremely improbable.

We are therefore driven to the conclusion that the deviation that occurred after the second operation was the result of afferent cerebro-cerebellar tonus impulses reaching the left half of the cerebellum from the ocular centres in the right hemisphere that had not been interfered with in the first operation. The insignificance of the deviation is accounted for by the fact that the chief ocular (frontal) area had been removed upon the right side, thereby cutting off most of the normal afferent cerebro-cerebellar impulses to the left half of the cerebellum for which of course partial compensation had been made. When, therefore, the right lateral lobe was removed, the eyes practically remained in the primary position just as they do in section of both vestibular nerves or section of all the nerves to the ocular muscles. In other words removal of areas of cerebral cortex upon one side tends to counterbalance the effects of removal of the lateral lobe of the cerebellum upon the same side, or of section of the vestibular nerve on the opposite side. Extirpation of one lateral lobe of the cerebellum, followed later, by removal of part of the frontal eye-area of the opposite cerebral hemisphere gave results like those observed when the cerebral ocular area was removed first and then the opposite lateral lobe of the cerebellum.

Extirpation of one lateral lobe of the cerebellum followed later by removal of part of the frontal eye-area in the cerebral hemisphere of the same side gave results, like those observed when the cerebral eye-area was first removed and then the lateral lobe of the cerebellum on the same side.

Removal of part of the frontal eye-area of one cerebral hemisphere and extirpation on the same side of the lateral lobe of the cerebellum in one operation caused the eye on the side of operation to turn toward the side of the lesions. The eye on the sound side was not appreciably affected.

These experiments seem to indicate that in the conjugate lateral movements of the eyes in the dog and cat the contra-lateral eye in its outward movement (external rectus) is more strongly represented in the cerebral cortex than the homo-lateral eye in its associated movement (internal rectus). Similarly in the cerebellum the outward movement of the homo-lateral eye (external rectus) is more strongly represented than the corresponding associated movement of the contra-lateral eye (internal rectus).

The deviations and strabismus that follow ablations of portions of the cerebellum are usually attributed to irritation of the remaining fibres or centres. But Risien-Russell considers them as paralytic phenomena because, after compensation, the deviation reappears in ether or chloroform narcosis, and no mere irritative effect could behave in this way. The results of Ferrier and of Luciani support this view. The question arises as to whether the effects of removal of portions of the cerebellum are due to the withdrawal of some direct influence of the cerebellum upon the muscles, or to an indirect effect upon the muscles brought about through the agency of the cerebral hemispheres of the same, or of the opposite side, viz., by the removal from the cerebellum of some influence originating normally in the cells of the cerebral cortex. Risien-Russell himself has shown that each half of the cerebellum exerts important influences upon the cortical cells of the opposite cerebral hemisphere, and that unilateral lesions of the cerebellum induce inequality of excitability in the two cerebral hemispheres, the hemisphere on the side opposite to that of the cerebellar lesion being the more excitable, although Luciani found increased excitability in some parts and depression in other parts. Risien-Russell believes the abnormal position of the eyes after ablation of parts of the cerebellum is due to withdrawal of cerebellar influence exerted directly upon the ocular muscles and not indirectly by way of the cerebral cortex. He bases his opinion upon the fact that after removal of part of one cerebral eye-area, ablation of the lateral lobe of the cerebellum upon the opposite side causes exactly the same amount of deviation that is seen upon removal of one lateral lobe of the cerebellum with the cerebral areas of the opposite side intact. He believes that spontaneous nystagmus is an irritation phenomenon reflexly induced, whilst the nystagmoid movements evoked by voluntary movements of the eyeballs is a paralytic phenomenon due to weakness of the muscles producing movement of the eyes in any one direction or to weakness of the antagonists of those muscles.

Beevor and Horsley[40] observed that nystagmus only infrequently occurred upon irritation of the cerebral cortex and then only in very limited areas anterior to the frontal ocular areas. In operations upon the cerebellum or upon its peduncles, Ferrier[8] observed that violent irregular nystagmus is always present during the manipulations and it is only some time after the eyes have quieted down that the nystagmus can be properly studied.

The spontaneous nystagmus which accompanies active or passive rotation is a highly co-ordinated act intended to facilitate the visual fixation of passing objects. Hence the short, rapid jerks which enable us to see the moving objects continuously without suspension of vision even during the adjustments of the eyes. Such adjustments of the eyes are not possible where the centres for the balanced cerebellar mechanisms are removed or impaired. We must conclude, therefore, that the spontaneous nystagmus observed upon removal of parts of the cerebellum is due to enhanced irritability relative or absolute of fibres or centres that are left intact. Thus in Risien-Russell's experiments, ablation of one lateral lobe caused lateral nystagmus with slow jerks toward the side of the lesion. As the centres for the lateral movements of the eyes are located just to the left and right of the pyramid in the dog, and in the right and left curve of the median lobe in the cat (Ferrier) it seems most probable that the operation for removal of the lateral lobe simply impaired somewhat the centres for one limb of the balanced mechanisms, thereby causing relatively increased tonus in the corresponding mechanisms of the opposite side and spontaneous nystagmus toward the side of the lesion.

Removal of both lateral lobes causes upward nystagmus with a tendency of the upper segment of each eye to rotate from within outward at first. This spontaneous nystagmus was replaced by nystagmoid movements which occurred only when the eyes were moved. The direction of these movements was that of the voluntary movements of the eyes.

By means of aural irrigations we have shown that bilateral irritation or depression of the labyrinthine receptors may be accompanied by vertical nystagmus. In other words equal irritation of corresponding cerebellar centres related to the horizontal semicircular canals was without effect in causing the usual lateral horizontal nystagmus which accompanies unilateral aural irrigations. We may therefore assume that in removal of both lateral lobes the centres for the lateral movements of the eyes on either side of the pyramid were equally affected, directly or indirectly. The possibility of such an occurrence is at once apparent when it is remembered that the centre for the vertical downward movement of the eyes in apes is in the posterior extremity of the vermiform process, whilst the centre for the corresponding upward movements is in the anterior extremity of the vermiform process. Bilateral irritation of the centres located in the lateral lobes (semilunar lobules) would account for the outward rotation of the eyeballs just after the operation.

Ablation of one lateral half of the posterior part of the middle lobe caused lateral nystagmus to the left and slightly upward whilst ablation of the whole posterior part of the middle lobe caused vertical nystagmus of an upward direction which is readily explicable when the functions of the centres located in that portion of the cerebellum are remembered. See Chapter XXII.

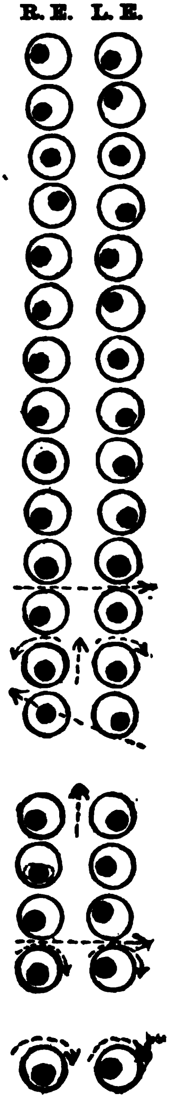
Ablation of the whole cerebellum causes no spontaneous nystagmus. There occur, however, in both eyes nystagmoid movements which follow the voluntary ocular movements. The results of this last experiment seem to support the view expressed in a previous chapter that removal or serious injury of any of the cerebellar centres causes, not spontaneous nystagmus, but nystagmoid movements and deviations. The significance of these movements should therefore be remembered in the diagnosis of cerebellar lesions. After compensation has been made for the nystagmus following cerebellar lesions the reappearance of the nystagmus under ether narcosis does not necessarily imply that the nystagmus was originally a paralytic phenomenon. Indeed the facts seem to justify the opposite conclusion, for such a highly co-ordinated act as physiological nystagmus necessarily implies intact mechanisms rather than mechanisms that have been more or less completely destroyed. However, as the centres in one portion of the cerebellum are set up against the centres in another portion (balanced mechanisms) it can be readily understood how impairment, destruction, or irritation of the centres in any one area may disturb the relations of the balanced mechanisms and so cause nystagmus. In these instances if the lesion of the cerebellum be a fairly gross one the ocular movements will be of the nystagmoid variety. With a lesser lesion they may resemble the movements of true spontaneous nystagmus, but some irregularity will be exhibited, especially in the reactions to aural irrigations, rotations, or galvanism.

In accounting for the discrepancies between the results of various physiologists it should be remembered that it is a matter of some difficulty to determine the direction of nystagmus in animals, and that in experiments upon the exceedingly sensitive labyrinth "retardation phenomena" may supervene, making it appear that similar operations give contradictory results. The rapidity of the short elements in nystagmus is so great that during their execution vision is impossible. For the same reason direct observation of these movements in the eyes is often an impossibility. Nystagmus is more conveniently studied in the human subject where the findings can be corroborated by the subjective sensations (i.e., sense of rotation, apparent movement of external objects) and above all by the aid of the ophthalmoscope.

The discrepancies between the observations of Risien-Russell and those of Luciani after ablation of one "lateral lobe" and "one half" of the cerebellum respectively can be accounted for by the fact that Luciani's operation removed the whole half of the cerebellum, including ocular centres in and about one side of the median lobe which of course escaped in Risien-Russell's operation.

These experiments demonstrate conclusively the existence of both cerebral and cerebellar balanced mechanisms for the control of ocular movements. In the dog and cat the only movements that seem to be represented in the frontal ocular areas are the lateral and upward vertical (Risien-Russell) whilst in the apes and presumably in man, the various other movements are represented. The cerebral mechanisms are represented in paths that lead from each frontal ocular area through the corona radiata, with or without interruption in the basal ganglia, to the oculomotor nuclei of the opposite side. The centres in each frontal area control the muscles that move the eyeballs in a certain direction and at the same time inhibit the antagonistic muscles which move the eyeball in the opposite direction. These mechanisms are of course under voluntary control, but perhaps owing partly to their relation to the cerebellar mechanisms the ordinary movements of the eyeballs are mainly executed in a reflex manner.

DIAGRAM TO SHOW THE EFFECTS OF THE VARIOUS OPERATIONS UPON THE CEREBELLUM, WITH THE RESULTS OF SOME OTHER OPERATIONS FOR COMPARISON.



1. Section of left middle peduncle in rabbit (Magendie).
2. Section of right vestibular nerve in sheep (Biehl).
3. Section of both middle peduncles (Magendie).
4. Incision of left lateral lobe (Gratiolet and Leven).
5. Ablation of left half of cerebellum (Luciani).
6. Removal of left lateral lobe; just after operation (Risien-Russell).
7. Ditto when effects of anesthesia have passed off (Risien-Russell).
8. Removal of both lateral lobes; middle lobe intact (Risien-Russell).
9. Excision of left half of posterior part of middle lobe (Risien-Russell).
10. Excision of whole posterior part of middle lobe (Risien-Russell).
11. Total ablation of cerebellum (Risien-Russell).
12. Ablation of left lateral lobe. Nystagmus (slow) to left (Risien-Russell).
13. Removal of both lateral lobes. Nystagmus at first; later nystagmoid movements (Risien-Russell).
14. Extirpation of left half of posterior part of middle lobe. Nystagmus (Risien-Russell).
15. Ablation of whole posterior half of middle lobe. Nystagmus (Risien-Russell).
16. Section of eighth nerve of right side (Risien-Russell).
17. Section of eighth nerve of right side (Bechterew).
18. Nystagmus just after section of eighth nerve (right) (Risien-Russell).
19. The same, two or three hours after operation (Risien-Russell).

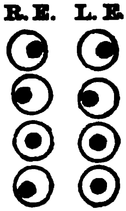
DIAGRAM TO SHOW THE OCULAR DEVIATIONS FOLLOWING REMOVAL OF PARTS OF THE OCULAR CEREBRAL AREAS AND OF PORTIONS OF THE CEREBELLUM.

a. Narcosis; eyes before operation.

b. Removal of frontal ocular area of right hemisphere.

c. Lapse of few days; eyes in normal position.

d. Removal of left lateral lobe of cerebellum.

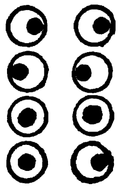


a. Narcosis; eyes before operation.

b. Removal of frontal ocular area of right hemisphere.

c. Lapse of few days; eyes in normal position.

d. Excision of right lateral lobe of cerebellum.

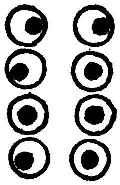


a. Narcosis; eyes before operation.

b. Removal of left lateral lobe of cerebellum.

c. Lapse of some days; eyes in normal position.

d. Removal of frontal ocular area of right hemisphere.

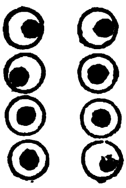


a. Narcosis; eyes before operation.

b. Extirpation of left lateral lobe of cerebellum.

c. Lapse of few days; eyes in normal position.

d. Removal of frontal ocular area of left hemisphere.



CHAPTER XXIII

ON THE OCCURRENCE OF NYSTAGMUS

Theoretically nystagmus can be produced only by interference with the co-ordinating mechanisms of ocular movement. As nystagmus is a highly co-ordinated movement effected by alternating reflexes in which inhibition plays such an important rôle, interference with the oculomotor nerves as in the case of the spinal motor neurones of which they are the analogues, does not cause true nystagmus but paralysis of ocular movement. And similarly, since, in the two sets of reflex arcs involved in nystagmus, interference with the efferent arcs of the final common path (ultimate neurone) upon one side of the body would not cause nystagmus, but rather absence of the related element of the nystagmus, or in other words a paralysis. For these reasons, and because inhibition is so frequently dependent on stimuli originating in the receptive field and transmitted by way of the afferent neurones (Sherrington[63]) we must look to the afferent arcs or co-ordinating centres in the cerebellum as the chief site of interference in the causation of nystagmus.

As these afferent arcs originate in the labyrinthine and retinal receptors, and possibly in portions of the cerebral cortex, it is evident lesions of any extent within the cranial cavity interfering with the afferent paths as well as lesions of very limited extent, e.g., a tiny patch of sclerosis, a small exudate, or a tiny thrombus or embolus in the cerebellar co-ordinating centres, may cause nystagmus. Hence nystagmus is set down in text-books as one of the general symptoms of disturbed cerebral function. The question arises, however, as to why nystagmus is so seldom produced by irritation or ablation of the cerebral cortex. The answer is that such nystagmus is occasionally seen (Beevor and Horsley[40]) although ordinarily it is inhibited by the conspicuous ocular deviations that accompany irritation and ablation of the cerebral ocular centres. It will be noted that these ocular deviations are of such direction as would inhibit the nystagmus that should accompany such a lesion, i.e., that they are in the direction opposite to that of the short elements.

Destruction of the cerebellum or lesions destroying or causing suspension of function of the ocular co-ordinating centres to any great extent causes, not nystagmus, but nystagmoid movements in which the sharp differentiation of the short and long elements is wanting.

Visible nystagmus most frequently results from lesion or interference with the ocular or labyrinthine mechanisms. Milder grades, however, readily detected by means of the ophthalmoscope, are of very frequent occurrence and are usually overlooked. These milder forms of nystagmus are generally caused by irritation of the cerebral cortex by toxines of various kinds circulating in the blood since the cortical centres seem to be more readily affected by various poisons than the purely reflex centres, cerebellar or other. On this ground may be explained the recurrence of ocular deviations in the experiments of Risien-Russell.[60]

These mild ophthalmoscopic forms of nystagmus are important features in minor forms of disturbances of equilibrium. There are good reasons for believing that vertigo, with its attendant subjective distress, is seldom or never experienced in the absence of nystagmus, although it has been recorded that some persons suffered from vertigo even after removal of both eyeballs. The close relation between vertigo and vomiting makes it probable that the vertigo of gastric disorders may not wholly originate in toxemia of the cerebral centres, but may be mediated through afferent impulses initiated by direct irritation of the receptors in the alimentary canal.

Miner's nystagmus seems to be caused by the strained and unusual position of the head, whereby certain sets of vestibulo- and oculo-cerebellar mechanisms are in constant and prolonged use, so that exhaustion of the neurones involved ensues with a condition of localized neurasthenia similar in every respect to an occupation neurosis. When the subject resumes his natural position the weakened portion of the oculo- or vestibulo-cerebellar mechanisms is no longer capable of offering the adequate counteraction to the opposing mechanisms to secure a proper balance of reflex tonus innervation. The result is nystagmus. Possibly a similar explanation, i.e., neurasthenia of certain sets of vestibulo-cerebellar mechanisms holds for Gertier's disease with its attacks of vertigo, paralysis (paresis) of the neck muscles allowing the head to fall forward, etc. This disease occurs frequently in France, Switzerland, and Japan, and is especially noted in those who are much fatigued and neurotic or emotional, and in those working in a lowered position (Starr[226]). The author has seen nystagmus occur in an infant of neurotic heredity upon the eruption of each of the first four teeth. The child was in fair general health, and fairly well up to the average in intelligence and development for children of her age. There was a distinct tendency for the tooth on one side to appear long before the corresponding tooth on the other side. With the eruption of the left, lower, central incisor, there was a deviation of the head toward the right, and a nystagmus of mixed type with a general direction toward the left. The child on looking at any object, held in a fixed position upon her left, lowered the head so that she could look at it out of the right corners of her eyes, a position in which the nystagmus was at a minimum. The explanation of the teething nystagmus is afforded by the lack of development of the cerebral (inhibiting) centres and by the instability of the cerebellar mechanisms which responded to unilateral impulses reaching it by way of the trigeminus and the nucleo-cerebellar tract. Such abnormal responses to more or less ordinary stimuli are not uncommon in neurotic individuals, and they may explain many forms of obscure vertigo as that from nasal polypus, contact of the skin with cold objects, e.g., the doctor's hand, etc. An interesting feature of this case was the fact that incision of the gum over an erupting tooth terminated rather suddenly an attack which, judging from previous experience, seemed destined to last for some weeks.

Another interesting case of nystagmus was seen in the first days of an attack of erysipelas starting in an infant of five months over the mastoid area just behind the left auricle. The nystagmus, which was of the mixed type, had a general direction toward the left and with the watch, and was undoubtedly due to heightened irritability of the ampullary receptors of the superior and horizontal canals caused by the local inflammation. The local application of ice checked the nystagmus and helped to soothe the severe gastric irritability. The child went on with the struggle which was a prolonged one. The disease spread all over the scalp and body with the formation of numerous abscesses, but eventually ended in complete recovery. Here again, perhaps, we must recognize lack of cerebral development coupled with cerebellar instability as the fundamental factor resulting in a manifest quantitative disproportion between effect and cause, the latter in this instance being of necessity a trivial difference of temperature in the two labyrinths aided perhaps by toxæmia. The nystagmus disappeared inside of forty-eight hours.

Nystagmus more frequently occurs in children than is usually supposed. Osler and Peterson[227] have reported three cases occurring in young children affected with diplegia.

It is possible to differentiate nystagmus of central cerebellar origin from that originating in labyrinthine disturbances. Nystagmus due to the latter has always a fast and a slow element, and the movements are executed about definite axes of the eyeball corresponding to rotations or movements in fixed and definite planes, whereas in cerebellar nystagmus, e.g., from a gross destructive lesion in the vermis, the movements of the eyeball are apt to be irregular in that they occur, not about any axis of the eyeball, but are more in the nature of irregular oscillations back and forth, or circumduction movements of the eyeball as a whole. There is an irregular inco-ordinated action of all or part of the muscles, but no set of opponents work together in such a way that the movements could be said to have a regular fixed direction as in true nystagmus. Moreover, voluntarily turning the eyes in a certain direction does not inhibit the movements as in labyrinthine nystagmus, though it may diminish them to some extent or develop abnormal movements. However, some cases of cerebellar nystagmus due in part to destruction and in part to irritation, may have a definite direction and may be exaggerated by turning the eyes in a certain direction, thus resembling labyrinthine nystagmus, but in the case of cerebellar nystagmus there is always some element which would be atypical for labyrinthine nystagmus as shown by the reactions to aural irrigations, rotations and galvanism. Thus in one case due to a syphilitic deposit in the vicinity of the vermis, there being no cerebral or cranial nerve symptoms, the nystagmus was of the vertical type and was not affected by lying down. On looking up, the eyes were steady, but on looking toward the feet, the eyeballs began to jerk upward toward the vertex which is opposed to all experience in the nystagmus of labyrinthine origin. With this nystagmus there was an "empty feeling" all over the body on the right side, but with no paralysis and no disturbance of the reflexes or loss of cutaneous sensation. There was slight inco-ordination in the right hand movements as compared with the left. Lying in bed with the eyes closed and directed toward the feet, there was experienced a sense of rotation in the mesial plane "occiput first." Sitting up with the eyes closed, there was experienced a sense of rotation about the long axis from right to left. The hearing was good on both sides, and there were no noises in the ears. All combinations of aural irrigations were tried, with the subject in the supine posture. Bilateral irrigations at 61° F. caused the eyes to jerk furiously both on looking up and down, whereas bilateral irrigations at 116° F. caused the eyes to jerk upward upon looking toward the vertex, but absolutely stopped all motion upon looking toward the feet. These symptoms and signs made probable the diagnosis of a lesion affecting the posterior portion of the middle lobe of the cerebellum and extending somewhat to the left from the middle line, the paresis in the muscles being in this instance in the inferior recti. The peculiar numb empty feeling on the right side of the body extended from the head to the foot. A similar feeling was noted in aural irrigations but it never extended much below the head and neck. This symptom seems to be characteristic of cerebellar disturbance and should be carefully studied in every case before being classed as merely one of the paræsthesiæ. Another curious symptom this patient presented was amenorrhœa for a period of one year. Her menstrual flow returned two weeks after the commencement of mercurial inunctions coupled with iodide of potassium internally. The amenorrhœa was probably only indirectly associated with the cerebellar lesion. Formerly the cerebellum was supposed by physiologists to be functionally related to the sexual organs. Ferrier[8] in his experiments found nothing to support such a view. In the present instance the amenorrhœa was merely the result of general ill-health and anæmia consequent upon the syphilitic infection.

The importance of the labyrinthine mechanisms in equilibration has long been recognized by physiologists. We have seen the scope and nature of some of their relations in this respect. We have also seen that they have important relations with ocular movements, and with circulatory and respiratory as well as with gastric and intestinal function. Their delicacy as evinced in the foregoing experiments, and the possibility of latent, hereditary, or acquired defect, coupled with their important and extensive relations, give these mechanisms a new importance in the etiology and treatment of neurasthenias, both local and general, as well as of the various neuroses, and especially those associated with disturbed gastric function. The behaviour of the stomach in rotations and in aural irrigations, points to these cerebellar and labyrinthine mechanisms as a possible factor in the etiology of hypertrophic stenosis of the pylorus in infants and adults.

The relations of the cerebellum to the cerebrum, are not so clearly defined, but there is enough evidence to show the existence of important afferent and efferent cerebellar paths between these two organs. This is a further reason for according the cerebellum and its mechanisms an important rôle in the etiology of neuroses and neurasthenias, in which hurry and restlessness, both physical and psychic, are such features. Migraine also is undoubtedly related to some defect in the cerebellar mechanisms, and the fact that it is said to yield to hypnotic suggestion, can probably be explained by the afferent and efferent relations between the cerebellum and the cerebrum. The importance of the afferent cerebellar paths, especially those associated with the eye and ear, should be constantly remembered. A trifling defect in either of these organs, although in time it may be compensated, may be sufficient to impair the automatic mechanisms to such an extent that the individual has to devote an undue amount of concentration, time, and energy, to the performance of ordinary acts. The result is a handicap for the individual, and perhaps impairment of the general health.

PART III

SEASICKNESS

CHAPTER XXIV

STUDIES IN SEASICKNESS

On December 27, 1908, observations of the circulation and blood-pressure were taken aboard the steamship *Taurus*. The round trip lasted about eight hours. The weather was mild, but there was sufficient rolling and pitching to affect subject "B" who was very susceptible, and yet not enough to interfere directly with the correct observation of the blood-pressure, or indirectly by necessitating balancing efforts on the part of the subject. See protocols at the end of Chapter XXIX.

Protocol 1 shows the results of observations made on this trip. After the start, there were signs of reaction on the part of the circulatory mechanisms resembling those seen after mild rotations and aural irrigations. The first portion of the protocol represents the condition of the circulation after the exertion and excitement of trying to catch the boat. After a time, however, the blood-pressure and pulse-rate fell steadily as the result of fatigue of the vaso-constrictor mechanisms (not well shown in the protocol because the blood-pressure was not taken sufficiently often) and of stimulation of the vagus centre. The slow pulse-rate shown between 10 a.m. and 11 a.m. is very unusual for this particular subject, and undoubtedly represents mild vagus stimulation. The recovery of the vaso-constrictor mechanism from 12.30 p.m. on, when the boat was at rest and the machinery stopped is worthy of note. The chief symptoms experienced by the subject were a slight headache, and the familiar "queer feeling all over" which was present only at times. In addition to these there were slight disturbances of the nervous system, as manifested by disinclination for work, irritability of temper, and a consciousness of the respiratory movements. Long, deep inspirations were a matter of frequent occurrence.

On April 4, 1909, another trip was made upon the steamship *Angler*. On this occasion frequent observations on the pulse-rate and blood-pressure were made. The trip lasted about seven hours and a half. The weather was mild, but there was enough motion to produce the subjective phenomena of seasickness in its early and milder phases. The nervous manifestations were headache, with fulness and lightness in the head, paræsthesiæ of the scalp, especially a sense of tension in the occipital region, psychic and motor depression, irritability of temper, disinclination for work, perversion of sensory function, whereby the respiratory and gastro-intestinal movements were registered in consciousness, perversion of the sense of smell, whereby the odour of tobacco-smoke, agreeable under ordinary circumstances, became obnoxious, photophobia, and annoyance from the use of the eyes, especially in looking at the moving water (fatigue of the oculo-motor apparatus).

The changes in the circulation, as shown in protocol 2, were not striking, nevertheless they show the characteristic reaction of the circulatory system to repeated irritation of the medullary centres, and though milder in degree, resemble those changes so constantly met with in rotations and aural irrigations. All through the trip the vaso-motor mechanism was in a constant state of activity in response to mild stimulation of the labyrinthine receptors. The movements of the boat were not of sufficient abruptness and range to cause overirritation with subsequent exhaustion. Hence the protocol shows rather an enhancement of the circulation, with a slowing of the pulse, the latter in great part the result of mild stimulation of the cardiac vagus centre. These observations show that even in the presence of unimpaired circulation, all the subjective phenomena of seasickness may be present. It follows, therefore, that the prime cause of seasickness is not impaired circulation, although profound changes in the circulation may, and usually do, occur in any given case.

Digestive disturbances appeared early. The "lump-sensation" in the stomach—i.e., a feeling as if a foreign body were in that organ, is one of the early symptoms of seasickness, just as it is in the sickness of rotations and aural irrigations. This symptom results in part from sensory perversion, and in part from overactivity of the muscles of the stomach, both of which are in turn to be attributed to irritation of the medullary and cerebral centres. On different occasions the stomach was evacuated and irrigated, to determine the cause of this "lump-sensation." In many instances a mass of mucus was recovered, but this, though of importance as a secondary factor, could not be considered the prime cause, as it frequently happened that no such disturbing element was to be found within the stomach, and where such was found, the symptom often persisted after its removal. In support of this view, may be cited the concomitant slowing of the pulse in seasickness and in rotation- and irrigation-sickness, as well as the vaso-motor changes, nausea, increased salivation, yawning, deep, sighing respirations, and other symptoms, all of which point to disturbed function in the medullary centres.

On May 30, 1909, further observations were made on the steamship *Angler*. The trip lasted about eight and a half hours, during which observations on the pulse-rate and blood-pressure were made almost constantly. The results are recorded in protocol 8. The weather was mild. There was rather more motion to the boat than on the previous occasions. An early start was made for the boat, so as to avoid the effects of rush and hurry upon the circulation. No food had been taken since the previous night, so that a test meal could be given, and observations made on the functions of digestion, etc. The figures recorded down to 10.24 a.m. show but little effect upon the circulation, and yet the subject experienced most of the symptoms of seasickness of mild grade, e.g., "lump-sensation" in the stomach, increased secretion of saliva, headache, fulness in the head, dizziness, pain in the eyes, disinclination for work or effort of any kind. The figures from 10.24 to 12.38 p.m. show practically the same thing, although there was considerable rolling of the boat during this period. The subjective symptoms were increased. Food was taken with the result that it subsequently caused pain in the stomach. Occipital headache and pain in the back of the neck upon the right side were features of this period.

The blood-pressure showed a slight downward tendency.

From 12.38 until 2, vasc-motor fatigue was in evidence, with a steady decline in blood-pressure. The pulse-rate was increased to meet the failure of the vaso-motor system. The subjective symptoms of this period included nausea, headache, paræsthesiæ of the scalp, irritability of temper, muscular weakness and tremors. Eructations were frequent. Between 2 and 3.35 there was general fatigue, and especially fatigue of the vaso-motor mechanism with the pulse-rate moderately increased. The subjective symptoms persisted, causing general wretchedness. About 3.50, there was a tendency to recovery on the part of the vaso-motor mechanism, with a drop in the pulse-rate. The general condition of the circulation was fair, and yet the subject's condition was far from good. The symptoms were: pain in the muscles of the back of the neck, upon the right side, extending up into the occiput, general weakness, and sweating, "lump-sensation," eructations, dull ache in the eyeballs. All through the trip the arteries were alternately contracting and dilating. The general effect upon the circulation was to induce fatigue, with a consequent fall in the blood-pressure. An Ewald breakfast was given subject "B," and allowed to remain in the stomach 1 hour and 54 minutes. The K I absorption test showed iodin in the saliva in 54 minutes. The motility test showed salol in the intestine in 2 hours and 2 minutes. The amount of chyme recovered was about ℥vi. This had to be computed, as the subject vomited on introduction of the stomach-tube. Analysis of the filtered gastric juice showed total acidity, .13% by weight; free HCl, .05%; combined HCl, .06%. A trace of blood was found in the chyme, as the result of straining and congestion during the vomiting. There was no other abnormality. Analysis after a test meal given to subject "S" on the same trip and retained 1 hour and 12 minutes showed: total acidity, .36%; free HCl, .26; combined HCl, .09, with the K I test positive in 12 minutes, and the salol test positive in 1 hour and 7 minutes. This subject, however, has little susceptibility—at least he is not affected in weather such as that which prevailed on this occasion. Moreover, he is by times a boatman and "follows the water" as a business.

Analysis of specimen taken May 30, 1909, from subject "B" aboard the *Angler*. Ewald meal taken on fasting stomach at 8.30 a.m. and withdrawn at 10.24. Amount of chyme, ℥vi. Resorcin test for free HCl positive, but feeble reaction:

Tot. acidity, 38, or .13% by wt.
Free HCl, 14, or .05% by wt.
Comb'd HCl, 18, or .06% by wt.
Total HCl, 32, or .11% by wt.
Acid salts, 6, or .02% by wt.
Lactic acid, absent.
Peptonizing enzymes, diminished.
Other enzymes, normal
Starch, absent.
Amylodextrin, absent.
Erythrodextrin, trace.
Maltose, present.
Bile, absent.
Blood, present in small amt.
Mucus, very small amount.

Analysis of specimen taken May 30, 1909, from subject "S" aboard the *Angler*. Ewald meal ingested at 8.30 a.m. on fasting stomach; withdrawn at 9.42. Amount of chyme ℥iii. Resorcin test for free HCl positive.

Tot. acidity, 100, or .36% by wt.
Free HCl, 72, or .26% by wt.
Comb'd HCl, 26, or .09% by wt.
Total HCl, 98, or .35% by wt.
Acid salts, 2, or .007% by wt.
Lactic acid (direct and ether), absent.
Enzymes, normal.
Starch, absent.
Amylodextrin, present.
Erythrodextrin, present.
Maltose, present.
Bile, absent.
Blood, absent.
Mucus, very little.

On May 31, 1909, a trip lasting one hour was made in the motor launch *Maggie*. Observations on the pulse and blood-pressure were made almost continuously throughout the trip.

Protocol 4 shows the results. The subject "B" was somewhat fatigued before the start. There was little in the way of a swell upon the water, and yet the boat pitched and rolled somewhat. The circulation all through the trip showed slight fluctuations, but the arteries nevertheless were constantly contracting and dilating. At times the blood-pressure was rather low, but this was in part due to previous fatigue. Even in the fresh breeze, there were some subjective symptoms, such as "lump-sensation" in the stomach, and a "queer sick feeling" in the head, as well as deep, long drawn breaths and eructations. These symptoms were present in spite of the fact that the subject was not feeling badly. In the second half of the trip, the pulse-rate showed a tendency to slowing, in spite of the low blood-pressure. Note at the commencement of the protocol, the effect of recumbency upon the circulation, when vaso-motor fatigue has supervened.

The conclusions are:

1. That in the beginning of seasickness, the blood-pressure may show slight changes, the effects in general being a slight rise.

2. That the arteries are constantly contracting and dilating, the tendency in general being toward a state of contraction.

3. That the pulse-rate varies somewhat with the contraction and dilatation of the arteries, the chief feature being a tendency to slowing, with some rhythmic irregularity.

4. That cerebral, gastric and other "dolors" occur simultaneously with unimpaired or even with enhanced circulation.

5. That digestive disturbances even in the milder phases of seasickness set in early, and form an important and constant feature in the train of phenomena.

6. That the phenomena of seasickness seem identical with those of rotation-and aural-irrigation-sickness.

7. That disturbed circulation is not the prime cause of seasickness.

8. That gastric and circulatory disturbances, however much they may contribute as secondary causes to the phenomena of seasickness, are not the prime cause, but are themselves, together with the psychic and motor depression and the other phenomena, the result of irritation of the medullary, cerebellar and cerebral centres.

CHAPTER XXV

FURTHER STUDIES IN SEASICKNESS DURING A TRANSATLANTIC TRIP

On June 26, 1909, a trip was commenced from New York to Glasgow, on board the steamship *Caledonia* of the Anchor Line. Observations of the circulation were made at frequent intervals during each day. The passage was not rough, there being just enough motion to afford an ideal opportunity of testing the prolonged effects of mild irritation upon the nervous, circulatory and digestive mechanisms. Protocol 5 shows the effects of the first day's sailing upon the blood-pressure and pulse. Almost immediately after the start, typical symptoms of mild seasickness set in. The boat started at 2.22 p.m., and by 8 o'clock the subject experienced the familiar "lump-sensation" in the stomach, increased salivary flow, perverted sense of smell (the odour of tobacco-smoke being offensive), occipital headache, especially in the region of the right mastoid and paræsthesiæ of the scalp (e.g., "cap" sensation). With these there were eructations and a considerable decline in blood-pressure, with increased pulse-rate. These phenomena are undoubtedly characteristic of seasickness, but there was practically no motion to the boat which could produce such symptoms, for we were scarcely out of the harbour and the sea was as calm as a lake. An explanation of the symptoms therefore has to be sought elsewhere. The subject was an old victim of the sea, having crossed the ocean about fifty times, and never once without suffering from seasickness in one form or another. On one occasion he had an experience which throws considerable light on the phenomena just detailed, He had been in the habit of crossing in the smaller boats of the Anchor Line, and had come to the conclusion that the equinoxes were not the most favourable times for going to sea. On this occasion, however, circumstances compelled him to cross in the beginning of April, and a cabin was secured in the steamship *Astoria*, which was a comparatively small boat. The date of sailing was fixed for Sunday, and on Saturday afternoon a visit was made to inspect the boat. It was a dismal, dreary day. Everything was hurry and bustle with the crew. Things were in a state of chaos on board, as might be expected. The stewards were scurrying about without uniforms, the decks were in disorder, and everything was so different from the conditions that obtain on the open sea, where orderliness, discipline, and white, spotless decks are the rule. The appearance of things was distinctly discouraging. This, coupled with the thought, that at that particular time of the year, a rough passage was to be expected, greatly depressed the prospective traveller. In such a frame of mind he went below, to inspect his cabin. The odour and gloom between decks overpowered him to such an extent that he became dizzy and nauseated, and experienced much of the distress that is associated with genuine seasickness. In fact at each step on the deck, the latter seemed to sink beneath his feet, as with a gentle rolling motion, and with all the tantalizing, sickening effects that such a motion can have upon the seasick brain. The subject left the boat. His seasickness persisted, however, for quite some time as he travelled homeward by street-car. It should be mentioned that although the subject is very susceptible to seasickness, he is not at all affected by the motions of trains or street-cars. The outlook for the journey was dismal. From past experience he knew that but little food would pass his lips during the coming eight days. However, he grimly determined to make the most of it, and so set about eating as much as he could conveniently digest, with the hope of storing his organs with an available supply of energy for the days of privation to come. Heavy meals were the order of the day even to the minute the boat sailed. Finally the journey was commenced, and now all the feasting was over, and nothing remained but to wait. The passage was a very rough one. Most of the passengers were dreadfully ill for days, but not this one, who, although slightly affected, never missed making an appearance at meal-time.

In fact it was the least disagreeable passage he had ever had. It should be stated, however, that the weather was very cold, and the subject's cabin well ventilated during the whole journey. This experience opens up the question of the influence of the imagination in the etiology of seasickness. Unquestionably the imagination plays an important part. Coupled with subconscious memories of past experiences, it may be a powerful factor, and is always an important secondary source of discomfort. It must not be considered, however, as the primary cause of seasickness. The phenomena of the early part of this first day's sailing on the *Caledonia* may, therefore, be attributed to fatigue and excitement backed by strong subconscious memories and by present impressions and associations. And here once and for all, impressionability is to be distinguished from fear, for in spite of his experiences of the sea, the subject was never afraid of seasickness and never "gave in" to it, as the expression goes.

After dinner, at 7 p.m., the absorption and motility tests were tried. A positive reaction for iodin in the saliva was found in 2 hours and 17 minutes, whilst salol was found to be present in the intestine in 2 hours and 22 minutes. Absorption evidently occurred only after food has passed into the intestine, for all through the tests, the salivary flow was free.

Toward night there were still present: headache, and "lump-sensation" in the stomach and throat. The circulation was very much depressed, on account of vaso-motor fatigue induced in part by heat and exhaustion, and in part by the motions of the ship. The behaviour of the circulation upon lying down as shown in protocol 5, where the pulse-rate suddenly makes an extensive downward excursion, whilst the blood-pressure makes a correspondingly sudden and extensive upward excursion, indicates exhaustion of the vaso-motor apparatus, the dynamics of the circulation, especially the return flow to the heart, being favoured mechanically in recumbency. Such variations of pulse-rate and blood-pressure when represented in charts, show divergent and overlapping curve loops which are common features of seasickness and of circulatory fatigue in general and which afford an important clue as to the method of treatment.

The urine during this day was rather heavy, the specific gravity at different times being 1.036, and 1.038, with a marked reaction for glycuronic acid. The amount passed was low, considering the liberal amount of fluid taken during the day. The warm weather and free sweating were factors in the diminished amount of urine, though the disturbed circulation undoubtedly played a part. On the day previous to sailing, the urine showed a specific gravity of 1.030 with no glycuronic acid or merely a trace.

During the first night at sea the subject slept fairly well but was disturbed by dreams.

June 27th.—The subjective symptoms were very mild on this day, and were perhaps to be attributed to the hot weather, and to general fatigue as much as to the motions of the boat. The digestive symptoms were those of the "lump-sensation" in the stomach and throat, which was present at various times during the day, and eructations. The absorption test showed iodin in the saliva in 21 minutes after the midday meal. The motility test was not tried, because there were still traces of salicyluric acid in the urine since the test of the previous day. Occipital headache was present, with a tired feeling about the eyes.

Protocol 6 shows the effects upon the circulation. The blood-pressure was rather low, even after a fair night's rest and the morning cold tub. The pulse-rate and blood-pressure indicate vaso-motor exhaustion, whilst the steady drop in the pulse-rate indicates, in the presence of low blood-pressure, irritation of the vagus centre.

The urine on this day was somewhat increased, the specific gravity at different times being 1.034, 1.030, and 1.028, with a less marked reaction for glycuronic acid.

June 28th.—On this day the notes make no mention of subjective symptoms. The urine had increased in amount, and was about the normal for this particular subject. The specific gravity at various times was 1.028, 1.024, and 1.020. A mere trace of glycuronic acid was present. Regular meals were eaten and caused no trouble. The bowels were constipated from the beginning of the journey, so that cascara sagrada was taken on retiring.

Protocol 7 shows a decided improvement in vaso-motor onus due, in part perhaps, to the effects of the cold lunge, and the general tendency of the vaso-motor system recover under the influence of cooler weather. The figures indicating the pulse-rates and blood-pressures, when represented in chart form, showed less divergence of the curve loops upon lying down. The blood-pressure was well maintained under the influence of improved vaso-motor tonus. The pulse-rate was uniformly low. The effect of sound, refreshing sleep is well shown, the pulse and pressure curves running along uniform and parallel lines.

June 29th.—This was a cold, foggy day. The ship commenced rolling during the night, and continued to roll all through the day. The result was a return of the subjective symptoms, viz., "lump-sensation" in stomach, fulness and lightness in the head, occipital headache, flushing of the face, aching and heaviness about the eyes, fulness in the ears, paræsthesiæ of the scalp, psychic and motor depression, and irritability of temper with a tendency to worry about trifles.

Protocol 8 shows the effects upon the circulation. There was a return of the vaso-motor exhaustion, which the cold plunge did not appreciably benefit. There was a return also of the diverging and converging curve loops, representing the pulse-rates and blood-pressures, notwithstanding the coldness of the weather, and a thorough evacuation of the bowels. The porthole was closed all day, but in spite of this fact the temperature of the air in the cabin was rather low.

After the midday meal, the absorption and motility tests were made. The salol and K I reactions appeared simultaneously in 2 hours and 16 minutes.

The urine was normal in amount and specific gravity, the latter being at various times 1.020, 1.022, etc.

June 30th.—On this day the ship rolled and pitched considerably, especially from the early afternoon on into the night. The subject felt very well in the forenoon, but as time went on, the symptoms of mild seasickness returned, with slight headache, and a feeling of dulness and heaviness. The "lump-sensation" was present in the stomach and throat, and with it a feeling of fulness and lightness in the head. At times there was a feeling as if "the top was being raised off the head." The saliva was increased, and the eyes ached. There was fulness in the ears, and a burning sensation in the stomach. The absorption test was tried after the midday meal, and iodin found in the saliva in 3 hours and 24 minutes.

At 6.30 p.m., dinner was over. There was considerable rolling and pitching during the whole evening. The motions, however, were slow and regular. The subject experienced no bad feeling, but gradually all the phenomena of disturbed digestion appeared, with increased secretion of saliva, "lump-sensation" in the throat and stomach, ringing in the ears, fulness in the head, etc. At 11 p.m., the contents of the stomach were removed, and that organ irrigated. A burning sensation, present before irrigation, persisted in spite of the latter. Analysis showed the presence of a moderate excess of mucus and of lactic acid with total acidity, .18% by weight, free HCl .13, and combined HCl .05.

Analysis of specimen taken June 30th from subject "B." Ingested at 6 p.m. a meal consisting of macaroni, brown bread with butter, 1 cup of tea, and some marmalade. Withdrawn at 11 p.m. Amount of chyme ℥iv, but a large amount was lost by vomiting. Resorcin test showed the presence of free HCl.

Tot. acidity, 52, or .18% by wt.
Free HCl, 38, or .13% by wt.
Comb'd HCl, 14, or .05% by wt.
Tot. HCl, 52, or .18% by wt.
Acid salts, ——.
Lactic acid (direct), present.

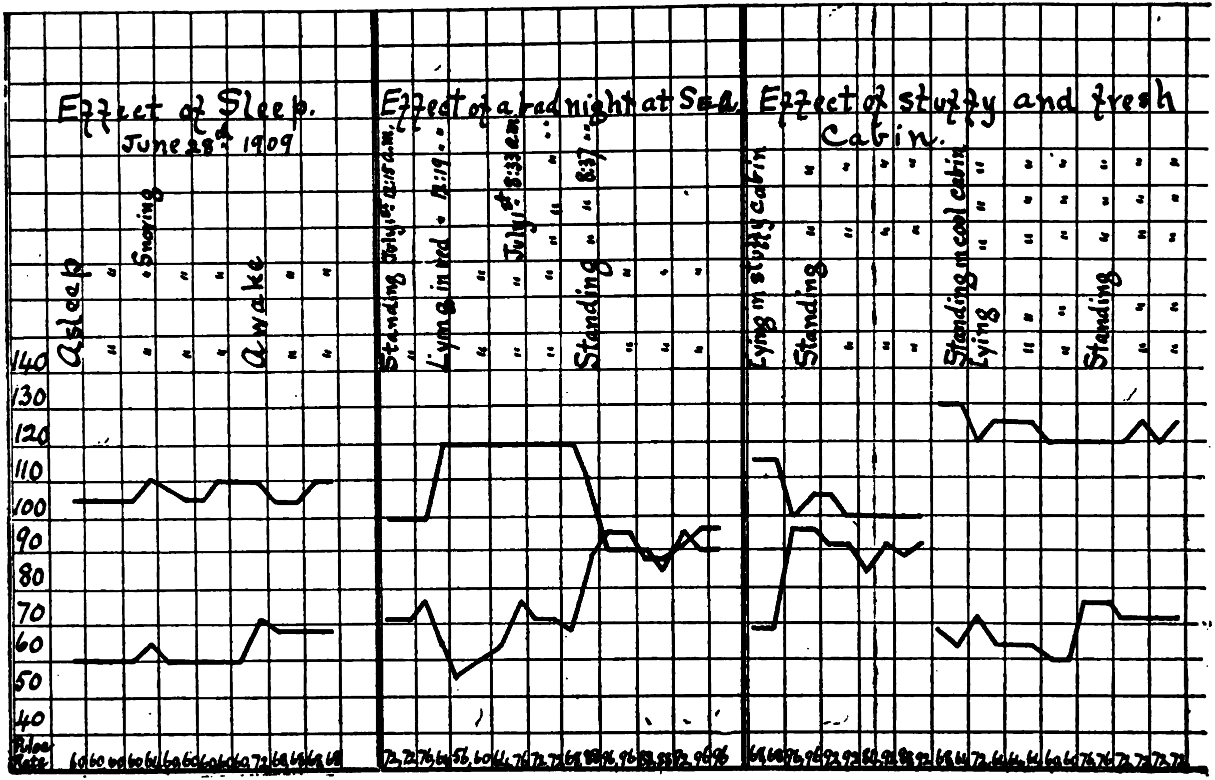
Starch, absent.
Amylodextrin, absent.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, present.
Bile, absent.
Blood, absent.
Mucus, moderate excess.

Absorption and motility tests not made.

The condition of the circulation during the day is shown in protocol 9. In the early part of the day the vaso-motor tone was fair, the response to the cold plunge being fairly good. As the day wore on, however, and especially with the onset of the subjective symptoms and of disturbed digestion, the vaso-motor mechanism showed evidence of fatigue, with lowered blood-pressure and increased pulse-rate.

The urine on this day was normal in amount. The specific gravity at various times was 1.018, 1.016, and 1.018. The subject was taking fluids freely. The weather was no longer very warm, consequently there was less sweating.

July 1st.—The boat rolled and pitched considerably



CHARTS ILLUSTRATING VARIOUS CONDITIONS IN SEASICKNESS.

Upper curves, blood-pressure; lower curves, pulse-rate. See protocols 9, 10, 24, 25.

throughout the night. It was interesting to find that even whilst the subject was lying in bed, the motions of the boat produced their usual characteristic effects in disturbing the organic functions, but, as in rotations and aural irrigations, these disturbances did not cause nausea, vomiting, and the usual extreme wretchedness, so long as the subject lay quietly in bed. All the subjective phenomena of seasickness were present on this day, the subject being too ill to take breakfast. Especially noticeable were the paræsthesiæ of the scalp over the right side of the occiput, and within the right ear in which there was a numb feeling as if there was an exudation within the tympanum.

Protocol 10 shows the extreme circulatory depression due to vaso-motor exhaustion, the result of rolling and pitching during the night. The vaso-motor system failed to respond properly to muscular exertion, e.g., chinning the bar. The respirations were increased to 24 per minute, doubtless as a compensation for the demoralized circulation and as a direct aid to the labouring heart.

At 10.18 a.m., an Ewald test meal was given on the fasting stomach. The absorption test showed iodin in the saliva in 28 minutes. The stomach contents were removed at 11.35 a.m. The amount of chyme recovered was ℥ii. It contained a considerable amount of thick, ropy mucus, and toward the end some pure blood. The stomach was irrigated when more mucus was removed and with it some more fresh blood. The analysis of the gastric contents showed: total acidity, .087% by weight; free HCl absent; combined HCl. .065.

Analysis of specimen taken from "B," July 1st. Ewald breakfast ingested at 10.18 a.m. Withdrawn at 11.35 a.m. Amount of chyme ℥ii. Evidence of gross blood. Resorcin test showed absence of free HCl.

Tot. acidity, 24, or .087% by wt.
Free HCl, absent.
Comb'd HCl, 18, or .065% by wt.
Tot. HCl, 18, or .065% by wt.
Acid salts, ——.
Lactic acid (direct), trace.

Starch, absent.
Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, present.
Bile, absent.
Mucus, considerable amount.
Blood, present.

The K I absorption test showed iodin in the saliva in 28

minutes. The salol motility test was not made. The urine was somewhat diminished on this day. The specific gravity was, at various times, 1.018, 1.016, and 1.010.

The day's experience shows how it is possible for an individual at sea to retire at night feeling in good general condition and to wake up in the morning with the circulatory and digestive mechanisms thoroughly demoralized.

July 2d.—This was a gloomy day, but the weather was not very rough. There was much rolling and pitching, however, and all the subjective phenomena of seasickness were experienced.

Protocol 11 shows the effects upon the circulation. Some observations upon the blood-pressure and pulse-rate of subject "S" are shown for comparison in protocol 11a. The circulation was similarly affected in both individuals. In both there was evidence of vaso-motor exhaustion.

Again it is to be noted that the cold tub had no lasting beneficial effects upon the vaso-motor mechanism, so that it is a question whether cold bathing is not harmful in these cases in which the vaso-motor system is exhausted from irritation of the medullary centres. Indeed subject "B" had known from long experience that the morning cold tub always seemed to aggravate rather than to help the subjective phenomena of seasickness. After the evening meal at 6.21 p.m., the absorption test was tried and iodin was not found in the saliva in 3½ hours, except for a fleeting faint reaction. However, the saliva reacted strongly for iodin on the next morning.

The urine on this day was normal in amount. The specific gravity at various times was 1.010, 1.018, and 1.020. At 4.16 p.m. the urine, with a specific gravity of 1.020, showed the presence of sugar. Only the test with Fehling's solution was tried, but using every precaution the conclusion was that the reducing agent was sugar. The previous meal was taken at 2.45 p.m., and consisted of pea soup, ℥vi, boiled codfish, ℥ii, potatoes, a large amount, one cup of coffee with milk and sugar, 2 currant cakes (sweet). At 5.50 p.m., the sugar had disappeared from the urine. This meal contained an unusual excess of sugar. The glycosuria was therefore in part at least of the alimentary type.

July 3d.—The weather on this day was rather mild. The ship, however, rolled and pitched considerably. All the subjective phenomena of seasickness were again experienced. Before breakfast the stomach was irrigated, and found to contain mucus and some blood. Protocol 12 shows the effects upon the circulation. There was marked vaso-motor exhaustion. Protocol 12a shows similar effects upon the circulation in subject "S." At 1.39 p.m. a meal was taken, consisting of consommé soup, ℥iv; roast mutton, about ℥ii; potatoes, a fair amount; water, ℥iii; sago pudding, ℥ii; 1 cup of coffee and 2 pieces of cake with sugar tops. The ship was rolling considerably and the "lump-sensation" was much in evidence. The subject slept for about 20 minutes during the afternoon. The effect of sleep is shown in the protocol, 2.31 p.m. to 2.51. At 5.35 p.m., the stomach contents were withdrawn. The amount of chyme recovered was ℥ii. It was sour smelling, and mixed with considerable mucus. Analysis showed: total acidity, .18%; free HCl, .09; combined HCl, .07. The motility test showed the presence of salol in the intestine in 1 hour and 25 minutes.

Analysis of specimen taken July 3d from subject "B." Ingested at 1.39 p.m. a meal as detailed above. Withdrawn at 5.35 p.m. Amount of chyme, ℥ii. Resorcin test showed the presence of free HCl.

Tot. acidity, 58, or .18% by wt.
Free HCl, 26, or .09% by wt.
Comb'd HCl, 20, or .07% by wt.
Tot. HCl, 46, or .16% by wt.
Acid salts, 12, or .04% by wt.
Lactic acid (direct), present.

Starch, absent.
Amylodextrin, absent.
Erythrodextrin, present.
Maltose, present.
Peptones, present.
Bile, absent.
Mucus, considerable amount.
Blood, trace.

The K I absorption test was not made. In subject "S" the salol test, which was tried by way of control, reacted in 2 hours and 40 minutes. The urine was normal in amount on this day. The specific gravity at various times was 1.024, 1.020, 1.022, and 1.006. There was neither sugar nor glycuronic acid present.

July 4th.—Moville was reached early on this morning. By 7 a.m., the Irish passengers had been sent ashore, and the ship was on its way to Glasgow. The weather was clear, but cold. There was a moderate amount of rolling.

Even on this day, some of the subjective phenomena of seasickness were present.

Protocol 18 shows the condition of the circulation. Evidently the vaso-motor system showed some evidence of recovery, for after the morning cold tub, there was a fair reaction, which persisted for at least half an hour. However, circulatory insufficiency appeared immediately when the least exertion was made, such as attempting to "pack up" to go ashore.

Although inactivity and hot weather may have been at times responsible for the conditions represented by the figures in the protocols, yet these could not have been the chief cause of the circulatory and digestive disturbances, as particular care was taken to see that the subject got sufficient exercise in the open air; and moreover, circulatory depression was present even when the weather was cold. The circulatory conditions on this day must, therefore, be attributed in part to the failure of recovery of the vaso-motor mechanism from the previous depression, and in part to the effects of the motion of the boat, and the jarring of the ship's machinery. It is interesting to note that very little motion or vibration suffices to protract the vaso-motor exhaustion, or rather to prevent recovery of the mechanisms.

At 6.58 a.m., an Ewald breakfast was given subject "S" by way of control. The stomach contents were removed at 8.10 a.m. The amount of chyme was ℥iii, with a moderate amount of mucus. Analysis showed: total acidity, .17% by wt.; free HCl, .11, and combined HCl, .04.

Analysis of specimen taken from subject "S" July 4th. Ewald breakfast ingested at 6.58 a.m., withdrawn at 8.10 a.m. Amount of chyme, ℥iii. Resorcin test showed the presence of free HCl.

Tot. acidity, 48, or .17% by wt.
Free HCl, 32, or .11% by wt.
Comb'd HCl, 12, or .04% by wt.
Tot. HCl, 44, or .15% by wt.
Acid salts, 4, or .01% by wt.
Lactic acid, mere trace.

Starch, absent.
Amylodextrin, trace.
Erythrodextrin, present.
Maltose, present.
Peptones, present.
Bile, absent.
Mucus, moderate amount.
Blood, absent.

Absorption and motility tests not made.

It is interesting to note that this individual's total acidity and free HCl were considerably reduced as compared with his normal condition, whilst the condition of his circulation was as much affected as that of subject "B," as shown in protocols 11, 11a, 12, and 12a. The circulatory mechanisms of subject "S" also showed on the slightest effort, marked exhaustion, which lying down did not seem to relieve as promptly as it did in the case of subject "B." From extensive and repeated observations of these two subjects, the conclusion was reached that one of them, viz., subject "B," the one on whom the observations were chiefly made during this voyage, possessed a keen and effective vaso-motor apparatus and a comparatively weak and irritable heart, whilst the other (subject "S") had a strong and efficient heart muscle, but a comparatively feeble vaso-motor mechanism. These facts were apparent in other ways. Thus in subject "B" cold air, or cold bathing had always a wonderful effect in bracing the circulation, except when the neuro-vascular mechanism was in a state of exhaustion from constant overstimulation, as occurred at times during the voyage. In subject "S" the effect of cold was to produce a chill, with blueness of the surface, and without any evidence of the tonic hyperæmia of the skin such as was always observed in subject "B." The inefficiency of the neuro-vascular mechanism in this individual "S" accounts for the fact that in him, as compared with subject "B," there was a less marked rise of blood-pressure noted upon lying down, although the pulse-rate fell, as in subject "B," to the normal. See protocols 11, 11a, 12, and 12a. It appears that this difference in the neuro-vascular mechanisms was in part at least, what rendered the one with the less efficient vaso-motor apparatus less susceptible to the effects of rotation and aural irrigation, and to galvanism over the mastoid areas. The effects of prolonged constant irritation of the medullary centres, such as occurs in conditions obtaining at sea, were sufficient to beget vaso-motor exhaustion, and when the latter supervened its effect upon the circulation was very marked, causing an almost continuous overlapping of the blood-pressure and pulse-rate curves. This difference in the sensitiveness of the neuro-vascular mechanism of different individuals, coupled with the varying sensitiveness of the labyrinthine receptors, throws considerable light upon the varying susceptibility of individuals to seasickness. The considerable rise in blood-pressure that, upon lying down, constantly occurred in subject "B" was due, no doubt, to increased blood-supply in the medullary centres procured, in the first instance, by recumbency which aided the return flow to the right heart. The improved circulation in the medulla, with the increased supply of nutrition, so enhanced the metabolic processes in the centres, that a certain amount of vaso-motor tonus resulted. Hence, in looking over the protocols, it will be seen that frequently the radial artery is in a more or less firm state of contraction with the subject in recumbency.

At 3.15 p.m. the passengers were landed at Stobcross Quay, Glasgow. The condition of the circulation immediately improved, as shown in protocol 18. There was evidence of improved vaso-motor tonus, though of course all the effects of vaso-motor exhaustion had not worn off.

CHAPTER XXVI

STUDIES IN SEASICKNESS (CONTINUED)

July 5th.—The general improvement of the circulation on shore is further shown in protocol 14. Improved vaso-motor tonus was manifested by the manner in which the circulation was maintained in the erect posture without undue overaction on the part of the heart. It was apparent, however, that the recovery of the vaso-motor mechanism was as yet by no means complete. At 7.58 p.m., the steamship *Tiger*, of Duke's Line, bound for Dublin, was boarded. Even before the boat started, there was evidence of loss of vaso-motor tone, due in part tc failure of recovery from the effects of the ocean voyage, and in part to the exertions made to catch the boat. Moreover, the cabin was rather warm. When the boat started, subjective phenomena soon appeared. The "lump-sensation" was in evidence, with fulness in the head, occipital headache, fulness in the ears, paræsthesiæ of the scalp, irritability of temper, worrisomeness, etc. The condition of the circulation during the journey is shown in protocol 14a. There was evidence of loss of vaso-motor tone. These observations show that after seasickness, the recovery of the circulation takes place slowly. They also show that it requires very little in the way of motion or jolting to cause a return of the malady with its vaso-motor exhaustion, and circulatory depression. Here it may be remarked that the vestibulo-cerebellar mechanisms of an individual may become accustomed to the motions of a large ship, so that going to sea on that particular ship or perhaps on another ship of equal size, would not occasion seasickness, whilst if such an individual, believing himself immune, were to ship in a smaller vessel, the difference in the rate and rhythm of the movements would be sufficient to cause profound illness. And such was actually the experience of a sailor who, upon first going to sea, shipped in a large boat. He went to sea for fully six months before he "got over being sick." After remaining with the same ship for fifteen years, he made a change and, shipping in a smaller vessel, found that he became seasick every time he went to sea. It took him several months for his vestibulo-cerebellar mechanisms to become attuned to the movements of the second vessel.

During this trip the weather was fair, but the boat being very much smaller than the *Caledonia*, and the rate and range of its movements so different, very little motion showed marked effects upon the labyrinthine mechanisms. Moreover, the jolting and vibration due to the machinery of the boat, were more in evidence.

The K I absorption test was tried at 9.15 p.m., and iodine was found in the saliva at 10.24 p.m.

On July 6th, at 7 a.m., the North Wall, Dublin, was reached, the condition of the circulation being as shown in protocol 14a. There is evidence of vaso-motor fatigue even after a fair night's rest.

July 10th.—On this day a trip from Dublin to Southampton was commenced on board the steamship *Lady Wolseley*, of the British and Irish Steam Packet Co. The boat started at 4.54 p.m. During the few days spent in Dublin, subject "B" indulged rather freely in alcoholic liquors. His chief drink was Bass's ale and Guinness's stout, although occasionally he took whiskey and soda. Up to this time no alcohol had been taken by him for over a year. Before boarding the boat he had taken, during the day, eight bottles of stout, and one whiskey and soda. Plenty of time was allowed to prepare for the journey, so that there should be no hurry, which might mar the observations on the blood-pressure. Just as the boat started, the circulation showed a marked degree of depression, as indicated in protocol 15a. This depression was chiefly the result of vaso-motor (and perhaps cardiac) inefficiency, due, in part perhaps, to the effects of alcohol. It is worthy of note that the pulse-rate did not fall when the subject lay down. This shows well the action of alcohol upon the cardio-vascular mechanism, and wherein it differs from the effects of mere fatigue, in which the heart enjoys considerable rest when the subject is in recumbency, a thing not permitted by an excess of alcohol circulating in the blood. The inference is, that whilst within limits, exertion may be a good thing for the heart, by stimulating the nutrition of its muscle, the kind of work thrown upon the heart by overindulgence in alcohol is never beneficial, but the reverse, as the rapid rate, even when resting in recumbency, does not permit sufficient time during diastole for the processes of proper metabolic interchange. These things, in addition to the direct effect of alcohol upon the heart-structure itself, lead to rapid deterioration and degeneration of the muscle.

During the early evening the water was calm, but about 9 p.m. the boat rolled and pitched somewhat. The "lump-sensation" in the stomach reappeared and with it dizziness, increased saliva, etc. The subject began to feel very tired, as people will who overindulge in alcohol. It was a great relief to lie down.

The circulation during this period of the journey showed fatigue of the whole cardio-vascular mechanism. The respirations were 24, with the subject feeling sleepy and very nervous.

July 11th.—During the night the weather became very rough. The ship rolled and pitched in an extraordinary fashion. Everything in the stateroom was hurled about, so that demoralization and disorder prevailed. When subject "B" awoke at 4 a.m., and whilst he still lay in bed, the motion of the ship made him dizzy and sick. He was lying with his head toward the bow, and with the right side next to the water in an outside berth. The "lump-sensation" in the stomach and throat was so marked, that he constantly kept "swallowing to keep it down." He experienced, in this position, a slight ache in the muscles attached to the occiput upon the right side. Any concentration or mental effort was disagreeable, and tended to make him sick. The subject lay upon either side to see if the position would have any effect in alleviating or aggravating his condition. It was noted that although lying upon the side made him feel worse than lying on the back, it did not make any material difference upon which side he lay.

By 8.15 a.m., the subject was suffering from all the effects of fully developed seasickness. The "lump-sensation" in the stomach was much in evidence. The stomach-tube was introduced, but nothing returned. Water,

℥vi, was now swallowed and the tube reintroduced. A mass of thick tenacious mucus was removed and with it some fresh blood. During all this time the blood-pressure was well maintained, partly on account of the muscular efforts made in balancing. At 7.30 a.m. an Ewald breakfast was given, after which the subject lay in bed, in order that he might retain it. During this time the ship continued to roll and pitch excessively. The subject experienced all the subjective phenomena of seasickness, with lump-sensation in the stomach, headache, nausea, increased salivation, psychic and motor depression, and that dreadful "dolor cerebri" familiar to all who have suffered from seasickness. He felt and acted as though he were being dragged to execution when told that it was time to remove the stomach contents, which was done at 8.30 a.m. It was a difficult matter to get the chyme through the tube, which was at first blocked by thick mucus with blood admixed. After much ado, ℥iv were removed. Then the stomach was irrigated, and more mucus, with a little blood, was removed. The subject stated that his stomach felt better after the irrigation. Analysis after the Ewald meal showed: total acidity, .06% by wt., free HCl absent, combined HCl, .02.

In subject "S," who vomited the contents of his resting stomach, analysis showed: total acidity, .04% by wt., with free HCl absent.

Analysis of specimen taken July 11th from subject "B." Ingested at 7.30 a.m., an Ewald breakfast. Withdrawn at 8.30 a.m.

Amount of chyme, ℥iv.
Reaction (litmus), faintly acid.
Free HCl (resorcin), absent.
Tot. acidity, 18, or .06% by wt.
Free HCl, absent.
Combined HCl, 12, or .02% by wt.
Acid salts, ——.

Lactic acid (direct), absent or mere trace.
Starch, absent.
Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Coagulating enzymes (chymosin, etc.), diminished or absent.
Coagulating zymogen (chymosinogen), diminished or absent.
Peptonizing enzymes (pepsin), diminished or absent.
Peptonizing zymogen (pepsinogen), diminished or absent.
Bile, absent.
Mucus, considerable excess.
Blood, present.
Absorption and motility tests not made.

Analysis of specimen taken July 11th from subject "S." Contents of resting stomach vomited during seasickness.

Reaction, faintly acid (litmus).
Free HCl, absent (resorcin).
Tot. acidity, 12, or .04% by wt.
Free HCl, absent.
Combined HCl, absent.
Lactic acid (direct), absent.
Starch, absent.
Amylodextrin, absent.
Erythrodextrin, absent.
Achroödextrin, absent.
Maltose, absent.

The amount of material vomited was about ℥ii, and consisted mainly of mucus.

In the early afternoon the sea became calm, and both subjects, after lying down all through the forenoon, were able to appear at table.

The condition of the circulation in subject "B" during this rough tilt is shown in protocol 15b. In recumbency the blood-pressure was higher than usual, due in part no doubt, to the muscular action incidental to tossing about in bed. In the erect posture it was difficult to determine the true state of the circulation, on account of the extreme rolling and pitching. Accurate manipulation of the instruments was hampered, whilst efforts at balancing caused sudden and frequent variations in blood-pressure. The blood-pressure figures must therefore be understood as representing in great part the effects of muscular exertion. At this juncture it was evident that the best time to study the circulation in seasickness is not during rough weather, but rather under milder conditions and in the lighter phases of the malady. At 3.25 p.m. Falmouth was reached. A pleasant stroll was enjoyed during a peaceful afternoon. The effects upon the circulation are shown in protocol 15b. There was improvement in the vaso-motor tone as evidenced by the well-maintained pressure in the erect posture with lowered, regular pulse-rate. The subject slept on the boat alongside Falmouth pier, and passed a fairly good night, but was disturbed early in the morning by the donkey engine unloading cargo.

July 12th.—At 9.18 a.m. the boat started for Plymouth. The weather was mild, and the boat quite steady. The subject felt well, and experienced little discomfort, excepting a slight occipital headache. The machinery, however, was responsible for much jolting.

Plymouth was reached about 3 p.m. During the afternoon, the subject strolled about the ancient town. At 6 p.m. the boat sailed for Southampton. The weather was calm, and beyond a slight occipital headache, with a little congestion of the face and head, the subject felt no disagreeable symptoms.

At 7.38 p.m., after dinner, the absorption test was tried and iodin was found in the saliva after 2 hours and 7 minutes. During this test the boat rolled and pitched somewhat, and subject "B" felt the "lump-sensation" and experienced a slight fulness in the head. He also had a transient attack of dizziness. The circulation was fairly well maintained, as shown in protocol 15c. The subject had moderately indulged in ale during the evening.

July 18th.—On awaking, the subject experienced slight symptoms of indigestion. The circulation showed evident signs of recovery, as seen in protocol 15d. The overlapping of the blood-pressure and pulse-rate curves upon first standing up, as seen at 5.57 a.m., illustrates an interesting feature in the physiology of the vaso-motor system. When an individual makes a muscular effort without thorough psychic concentration, the neuro-vascular apparatus may be slow in responding to the demands of the body for higher blood-pressure. The result is that the heart has to make up with increased rate for the tardy vaso-motor action. It has frequently happened with subject "B" that in walking up a series of flights of stairs in a half-hearted way, but nevertheless at a fair pace and without stopping, the heart-rate was enormously increased, and the breathing deep and laboured, whereas, when he roused himself thoroughly and bent himself to the task of running up the same stairs at a brisk trot, lifting his feet neatly and giving free play to the muscles of his whole body, the heart-rate was only moderately increased and the auxiliary circulatory function of the respiratory mechanism was not sc much invoked. Tardiness or lethargy on the part of the neuro-vascular mechanism is common in many diseased conditions. It is the direct result of toxæmia in infectious diseases, and it is to its effects in counteracting this lethargy of the reflexes that cold bathing with friction owes its value in these conditions.

Lethargy of the vaso-motor mechanism, and of the reflexes generally, is also met with in other than diseased conditions, e.g., in the waning period of the athlete's career when he has to "warm up" before he can attain his normal speed and accuracy of movement.

As the subject dressed, he felt weak and nauseated. His condition was attributed more to the stuffy cabin and the ale he had taken on the previous night, than to the effect of the boat's motion. As time wore on, the feeling passed off, and he felt very well as the boat glided up Southampton Water. At 6.10 a.m., Southampton was reached.

At 11.50 p.m., the first of a series of trips upon the steamship *Southwestern*, running between Southampton and Cherbourg, was commenced. The English Channel has a world-wide reputation for causing seasickness, and it was intended to make the most of every opportunity.

July 14th.—At 12.24 a.m. the boat started. The condition of the circulation before and after the start is shown in protocol 16a. There was evidence of recovery of the circulation from the effects of the previous journeys. The weather was mild, and the water in a still calm. The subject's berth was situated about amidships, and transverse to the long axis in such a manner, that his head was located near the middle of the boat, with his feet directed to the port side. On awakening at 6.30 a.m., the boat was pitching furiously, with a short, snappy motion combined with a slight spiral twist. The subject woke up with all the symptoms of seasickness, viz., "lump-sensation" in the stomach, with nausea and increased salivation. He was very nervous and apprehensive. Lying on the back gave most relief, whilst lying upon either side seemed to make him sicker. This will be readily understood when it is remembered that the chief motion of the boat consisted in pitching, which affected chiefly the horizontal semicircular canals in the position in which he lay.

It will be remembered that the sheep, with the vestibular nerve divided, lies upon the side of operation. In this position the muscles on that side are relaxed, and receive little or no cerebellar tonus innervation. Since these muscles are related to the labyrinth on the opposite side, i.e., of the sound side, the vestibular mechanism on that side may be said to be quiescent and at rest. The consequence is, that the muscles of the sound side require little or no vestibulo-cerebellar tonus to counterbalance their relaxed opponents. Practically no demand for tonus impulses is therefore made on the peripheral vestibular apparatus on the side related to these muscles, viz., the side on which the vestibular nerve has been divided. The result is, that the animal can maintain this position without suffering the torments of vertigo. In any other position there would be a demand for vestibular tonus-impulses from the side of operation and failure to supply them would effect a break in the balance, with disturbances of equilibrium, vertigo, nystagmus, etc.

The case of subject "B" is somewhat analogous. Turning upon one side sets at rest the labyrinth of the opposite side. Under the influence of active stimulation of the ampullary receptors on either side by means of the boat's motion, this position is by no means one of stable equilibrium, for as the boat pitches, more or less reflex muscular activity is evoked, with resulting tendencies to disturb the equilibrium. The fact that the muscles of one side have a tendency to be relaxed and at rest by lying on that side, whilst their opponents under the conditions of active labyrinthine-stimulation are not permitted to be at rest, only tends all the more to cause a disturbance of balance between the opposing muscles on either side. Under these circumstances, the effect of the boat's motion is to keep up a constant disturbance of the vestibulo-cerebellar balanced mechanisms, with resulting discomfort and distress to the individual.

At 7.15 a.m., the tube was passed, but nothing was recovered from the stomach. Irrigation of that organ was then practised, and a quantity of thick mucus, mixed with some fresh blood, was removed. The stomach felt better after irrigation. The condition of the circulation during this period is shown in protocol 16a. The blood-pressure was well maintained, but this was in part due to the muscular efforts of balancing. The vaso-motor tone was still deficient, as evidenced by the increased pulse-rate necessary to maintain a fair blood-pressure. At 7.20 a.m., the boat arrived at Cherbourg.

On the evening of July 14th the absorption test was tried on shore after dinner. The saliva reacted for iodin in 1 hour and 10 minutes. The subject "B" had some symptoms of indigestion, and was very nervous. The night was close, and he slept but little.

July 15th.—After dinner the absorption test was again tried. Iodin was found in the saliva in 1 hour and 35 minutes.

July 16th.—On this day an attempt was made to study the immediate effect which travelling on a moving train has upon the circulation. All that could be ascertained with any certainty, was that the peripheral blood-vessels were constricted at times, and the blood-pressure raised. Such results as could be fairly ascertained are shown in protocol 16b.

July 17th.—On this day the absorption test showed the presence of iodin in the saliva in 28 minutes.

July 18th.—At 2 p.m. lunch was taken, consisting of cold roast beef, potatoes, vegetables, bread and butter, and coffee with milk and sugar. At 7.20 p.m. the stomach contents were removed and the stomach thoroughly irrigated. At 7.50 p.m. an Ewald test meal was given. The absorption test was tried, and iodin appeared in the saliva in 10 minutes. With the motility test, no evidence of salicyluric acid was found in the urine as late as 9.22 p.m. At 9.05 p.m. the stomach contents were withdrawn. The amount of chyme removed was ℥ii. Analysis showed for the first specimen, i.e., the one taken at 7.20 p.m., or 5 hours and 20 minutes after a meat lunch, total acidity, .219% by wt.; free HCl, .138; combined HCl, .07; and for the specimen after the Ewald meal: total acidity, .188%; free HCl, .080; combined HCl, .051. It was found that notwithstanding the irrigation some meat from the midday meal had remained in the stomach during the Ewald meal.

A test meal was given subject "S" by way of control. In his case the analysis showed: total acidity, .299% by wt.; free HCl, .21; combined HCl, .07, with the absorption test showing the presence of iodin in the saliva in 15 minutes. The motility test was not tried.

Analysis of specimen taken July 18th from subject "B." Ingested at 2 p.m., lunch consisting of cold meat, lettuce, carrots, potatoes, coffee with milk and sugar, and bread with butter. Withdrawn at 7.20 p.m. Amount of chyme, ℥iv. Resorcin test showed the presence of free HCl.

Tot. acidity, 60, or .219% by wt.
Free HCl, 38, or .13% by wt.
Comb'd HCl, 20, or .07% by wt.
Tot. HCl, 58, or .20% by wt.
Acid salts, 2, or .007% by wt.

Lactic acid (direct), present.

Starch, absent.
Amylodextrin, absent.
Erythrodextrin, absent.
Achroödextrin, absent.
Maltose, trace.
Peptones, present.
Bile, absent.
Mucus, minute amount.
Blood, absent.

Absorption and motility tests not made.

Note. The chyme contained particles of lettuce and carrots and some small pieces of meat, but otherwise it was fairly normal.

Analysis of specimen taken July 18th from subject "B." Ingested at 7.50 p.m. an Ewald test meal. Withdrawn at 9.05 p.m. Amount of chyme, ℥ii. Resorcin test showed the presence of free HCl.

Tot. acidity, 38, or .13% by wt.
Free HCl, 22, or .08% by wt.
Comb'd HCl, 36, or .13% by wt.
Acid salts, 2, or .007% by wt.

Lactic acid (direct), trace.
Starch, absent.

Amylodextrin, absent.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, present.
Bile, absent.
Mucus, very little.
Blood, absent.

Analysis of specimen taken July 18th from subject "S." Ingested at 7.50 p.m. an Ewald meal. Withdrawn at 9 p.m. Amount of chyme, ℥iii. Resorcin test showed the presence of free HCl.

Tot. acidity, 82, or .299% by wt.
Free HCl, 58, or .211% by wt.
Comb'd HCl, 22, or .077% by wt.
Tot. HCl, 80, or .288% by wt.
Acid salts, 2, or .007% by wt.

Lactic acid (direct), trace.

Starch, absent.
Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, present.
Bile, absent.
Mucus, very little.
Blood, absent.

July 19th.—The condition of the circulation on this day is shown in protocol 17. The vaso-motor system had evidently recovered from the effects of the sea.

July 20th.—On this day, a trip was made on the steamship *Cygne* from Carteret to Gorey in Jersey, Channel Islands. The weather was pleasant, but there was a considerable roll on the *Manche*, and many passengers were made ill. An Ewald test meal was given on the fasting stomach to subjects "B" and "S." Both were seated in the hot sun, fairly well amidships, where all sorts of disagreeable sights and odours prevailed. The boat rolled and pitched very much. Subject "B" experienced only the faintest symptoms of seasickness.

The amount of chyme recovered from the test meal given subject "B" was ℥iii. Analysis showed: total acidity, .146% by wt.; free HCl, .06; combined HCl, .06. A trace of blood was found in the chyme. The absorption test showed the presence of iodin in the saliva in 15 minutes. The salol test showed the presence of salicyluric acid in the urine in 35 minutes. There was reason to suspect that this early reaction was the result of the salol taken on the previous evening. The stronger reaction appeared in 1 hour 20 minutes. The amount of chyme recovered after the test meal given to subject "S" was ℥iii. Analysis showed: total acidity, .146; free HCl, .153; combined HCl, .109. The absorption test showed a positive reaction in 14 minutes. The salol motility test reacted in 1 hour and 19 minutes.

Analysis of specimen taken July 20th on the *Cygne* from subject "B." Ingested at 8.14 a.m. an Ewald breakfast. Withdrawn at 9.20 a.m. Amount of chyme, ℥iii. Resorcin test showed a feeble reaction for free HCl.

Tot. acidity, 40, or .146% by wt.
Free HCl, 18, or .065% by wt.
Comb'd HCl, 18, or .065% by wt.
Tot. HCl, 36, or .130% by wt.
Acid salts, 4, or .014% by wt.

Lactic acid (direct), faint trace.
Starch, absent.

Amylodextrin, absent.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptonizing and coagulating enzymes, normal.
Peptones, present.
Bile, absent.
Mucus, considerable amount.
Blood, trace.

Analysis of specimen taken on July 20th on the *Cygne* from subject "S." Ingested at 8.16 a.m. an Ewald breakfast. Withdrawn at 9.25 a.m. Amount of chyme, ℥iii. Resorcin test showed the presence of free HCl.

Tot. acidity, 72, or .262% by wt.
Free HCl, 42, or .153% by wt.
Comb'd HCl, 30, or .109% by wt.
Tot. HCl, 72, or .262% by wt.
Acid salts, ——.
Lactic acid (direct), absent or faint trace.

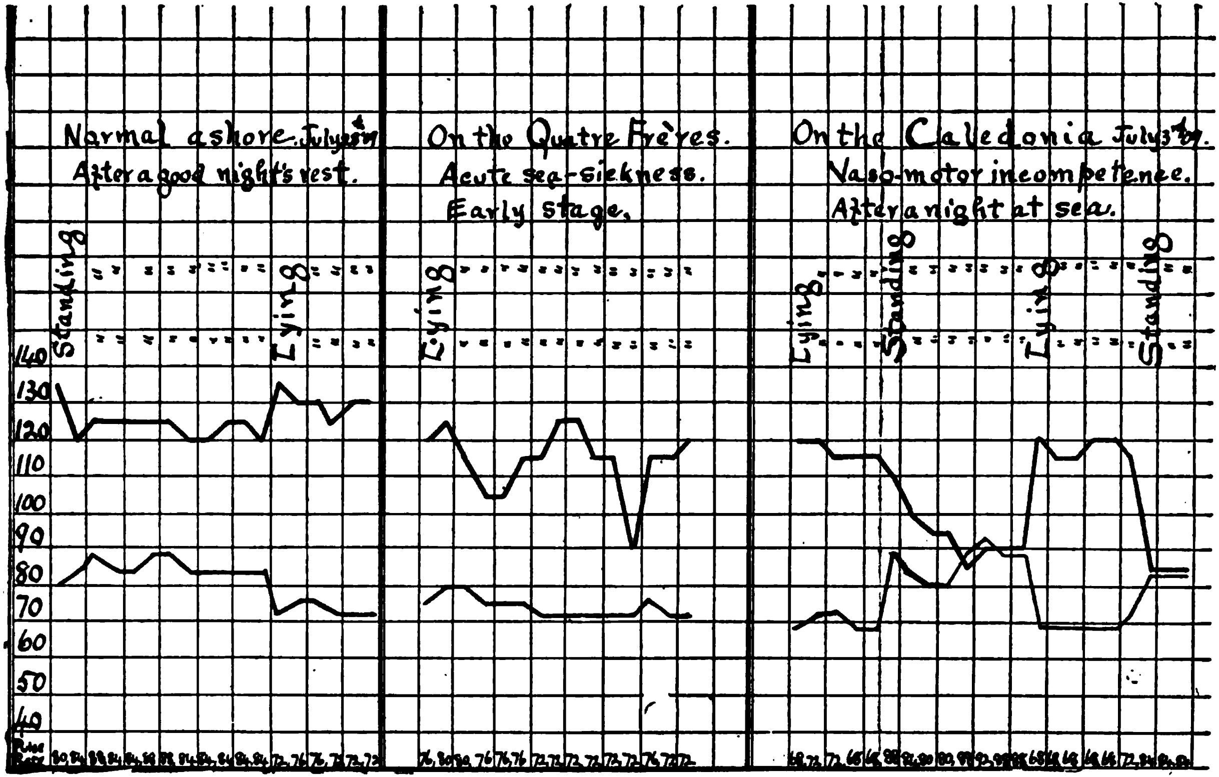
Peptonizing and coagulating enzymes, normal.
Starch, absent.
Amylodextrin, present.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, present.
Bile, absent.
Mucus, very little.
Blood, trace.

***Note.* A little gross blood appeared during the manipulations in evacuating the stomach contents. The blood was kept away as well as possible from the chyme.**

A comparison of the analysis from the test meals taken on the *Cygne*, and those taken on the previous evening shows that in the matter of acidity in general there was not much difference. Evidently both subjects had developed a certain amount of immunity for seasickness. It should be mentioned that the *Cygne* was in size and other respects very similar to the boats on which the subjects had been recently travelling.

The condition of the circulation during this trip is shown in protocol 17. Evidently the vaso-motor system was in keen working order. A fair amount of muscular action, however, was necessitated by the motion of the boat, and moreover the observations upon blood-pressure were taken in the open air.

July 22d.—On this day a trip was made in the fishing smack *Quatre Frères*. This was a sail-boat, and of course very much smaller than the boats in which the subjects had been recently travelling. From the very start it rolled and twisted and pitched in a fearful manner. Test meals were given to subjects "B" and "S," but "S" vomited his meal 26 minutes after its ingestion. It was only by hugging the floor of the cabin where he had wedged himself in between some cleats and the side of the vessel, that subject "B" succeeded in retaining his meal for the accustomed hour. The day was clear, but the



CHARTS ILLUSTRATING VARIOUS CONDITIONS IN SEASICKNESS.

Upper curves, blood-pressure; lower curves, pulse-rate. See protocols 19, 18, 12.

Manche was living up to its reputation. On this occasion subject "B" experienced all the horrors of seasickness in its most aggravated form. The odour of the cabin, not too wholesome at any time, became intolerable. When the time came for evacuating the stomach contents, all that the subject had to do when the basin was in place, was simply to turn on the side, and with one sudden act of vomiting, everything left the stomach. Water was subsequently taken, in order to clean out the stomach as much as possible. This was also immediately rejected, bringing with it nothing but mucus. The amount of chyme recovered was ℥xi. It was now 10.18 a.m. The boat had been out since 8.53 a.m., and was not expected to return before 6 or 7 p.m. On this account it was decided not to try the effect of drugs until the afternoon. Subject "B" felt much relieved for having his stomach emptied and irrigated. After a time, however, his wretchedness returned, and he was obliged to take to the floor again and wedge himself in an angle as best he could to keep from being tossed about. By this time he was very irritable, and the swirl of the water rushing past the side of the boat was extremely annoying. However, there was nothing left for him but passive resistance, which, in the sick and helpless, often represents the very acme of courage. He had made up his mind that the struggle was to be a long one, and bowed to the inevitable. At 12 m., however, it was an agreeable surprise to find that the boat was "alongside the wharf" and everything ready to go ashore. On inquiry it was learned from the skipper, that owing to the rough weather, the fishing nets had been swept away, and it was considered too risky to remain out longer.

The condition of the circulation was studied with great difficulty on this trip, and only at irregular intervals. The results are shown in protocol 18. There was a good deal of muscular activity at all times when the blood-pressure was taken. When the subject was lying quietly, if that were possible at any time, there were manifest signs of profound circulatory depression. In the afternoon subject "B" felt exceedingly nervous and irritable. It was only after several hours in bed that the effects of his rough tilt with the sea wore off. He had a slight headache, and every fibre in his limbs and body was in a tremour. Even a two hours' sleep did not relieve his nervousness. The "lump-sensation" in the stomach, which made its appearance upon the boat, remained with him as late as 5 p.m. The blood-pressure was well maintained, however, but the vaso-motor system was evidently still demoralized. Analysis after the test meal showed absence of HCl, both free and combined, and a total acidity of .021% by wt. Tests were made for the presence of the gastric enzymes, but an unfortunate accident destroyed the specimens before the customary time had elapsed. It was ascertained, however, that in the case of chymosin (rennet) coagulation was retarded considerably. It may, therefore, be assumed that the ferments were diminished. Both the K I and salol tests were negative, not only during the trip, but all through the afternoon. It was evident that no absorption had taken place, and that nothing had escaped into the intestines.

Analysis after the test meal given subject "S" and retained 26 minutes, showed total acidity .048% by wt., with HCl both free and combined absent. The K I and salol tests were negative during the trip and all through the afternoon.

The first urine passed by subject "B" after leaving the boat, was in amount ℥v, and slightly turbid, due to the presence of an excessive amount of mucus. The specific gravity was 1.028. There was no evidence of albumin or of sugar. Glycuronic acid was present.

Analysis of specimen taken July 22d on the *Quatre Frères*, from subject "B." Ingested at 9.10 a.m. an Ewald breakfast. Withdrawn (vomited) at 10.10 a.m. Amount of chyme, ℥xi. Reaction faintly acid (litmus). Free HCl (resorcin), absent.

Tot. acidity, 6, or .021% by wt.
Free HCl, absent.
Comb'd HCl, absent.
Tot. HCl, ——.
Acid salts, ——.
Lactic acid (direct), trace.
Starch, absent.
Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Coagulating enzymes, diminished.
Peptones, faint trace.
Bile, absent.
Mucus, abundant.
Blood, absent.

Analysis of specimen taken July 22d on the *Quatre Frères*, from subject "S." Ingested at 9.10 a.m. an Ewald breakfast. Withdrawn (vomited) at 9.36 a.m. Amount of chyme, ℥viii. Reaction faintly acid (litmus). Free HCl absent (resorcin).

Tot. acidity, 12, or .043% by wt.
Free HCl, absent.
Comb'd HCl, absent.
Tot. HCl, ——.
Acid salts, ——.
Lactic acid (direct), trace.
Starch, absent.

Amylodextrin, present.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, trace.
Bile, absent.
Mucus, considerable amount.
Blood, absent.

July 23d.—At Cherbourg the steamship *Southwestern*, bound for Southampton, was boarded at 10.12 p.m. At 11.47 p.m. subjects "B" and "S" received each, hypodermatically, gr 1/75 of atropin sulphate, and gr 1/40 of strychnin nitrate.

July 24th.—At 12.05 a.m. the boat started, and at 12.34 a.m. an Ewald test-meal was given to each subject. The weather was very rough. The boat pitched, twisted, and rolled badly. On this occasion the party travelled in the second cabin, as the sleeping compartment was situated in the bow where the motion was certain to be at its worst, and where one would be most liable to be made seasick. The berths were ranged around the sides of the ship as in a dormitory. The boat had scarcely started, when the passengers began to climb into their beds, and in a very few minutes no one was astir but the members of our party. Here an amusing incident occurred which those who practise psychic therapeutics will appreciate. The boat had been behaving badly, and there was every indication that there were rough times ahead. Suddenly the steward, a good-natured fellow, appeared with an armful of large white basins made of agate or papier mâché. One of these was placed beside the head, you might say under the nose of each passenger in a smart, businesslike way, as much as to say, "There! now it's up to you." The steward made the round of the compartment in quick time, but he had scarcely placed the last basin, when almost every one in the place commenced vomiting. He even had the temerity to place basins in front of subjects "B" and "S" and as he did so, he leered at them out of the corner of his eye, to see how they were standing the pace. It was easily understood why, for sanitary and economic reasons, vessels should be placed convenient to the passengers, but it was not at the time so apparent why the steward should so jauntily rattle his basins as he placed them around. His conduct seemed more intelligible the next morning when it was learned from stickers placed conspicuously in various parts of the boat that a certain remedy was a sure cure for seasickness and that it could be had at any time from the steward, price two shillings and nine pence. The test meals were eaten with relish, and although the boat pitched and rolled very badly, no ill effects were felt. In the stomach there was a cool, agreeable sensation, as though one had just eaten ice-cream. No perversion of the sense of smell was experienced. There was a peculiar sour taste in the mouth as though one were eating unripe apples. The saliva was not free, and yet the mouth was not dry. Both subjects felt an inclination for work. The balancing movements necessitated by the rolling of the ship were effectually and easily executed, without the usual anxiety and awkwardness. The subjects sat, stood, and walked about at various times during the hour that the test meals were retained. They felt exceedingly well during all the tossing and tumbling of the boat. At times subject "B," who is extremely susceptible to seasickness, felt a slight transitory fulness in the head. There were no symptoms referable to the stomach, in fact an agreeable feeling was associated with that organ. There was no psychic depression or distress. At 1.07 a.m., subject "B" still felt well, but on standing up was almost thrown by the rolling of the boat. Although feeling well he was disinclined to stand up or walk about. At this time there was a slight intimation of the "lump-sensation" in the stomach, though no disagreeable feeling was associated with it.

When the boat gave an unusually heavy lurch, both subjects felt it affect their heads momentarily, but no worry or distress was induced. At 1.33 a.m., subject "B" experienced a pronounced "lump-sensation" with a slight sense of nervousness or tremour in the muscles. Subject "S" felt no lump-sensation in the stomach at any time.

At 1.50 a.m., the stomach contents of subject "B" were removed. The amount of chyme obtained was ℥iv, A little blood also came with the chyme. Analysis showed: total acidity, .075% by wt.; combined HCl, .051, and free HCl absent.

The tests for enzymes showed diminution or absence of these bodies.

The K I absorption test showed the presence of iodin in the saliva in 11 minutes. The salol test was negative A faint reaction occurred in 50 minutes, but was of too doubtful a nature to afford basis for a conclusion as to stomach motility.

Analysis of specimen taken at 1.50 a.m., July 24th, on the steamship *Southwestern* from subject "B" after hypodermic injection of strychnin nitrate, gr 1/40, and atropin sulphate, gr 1/75. Ingested at 12.34 a.m. an Ewald test meal. Withdrawn at 1.50 a.m. Amount of chyme, ℥iv. Reaction to litmus, acid. Free HCl absent (resorcin).

Tot. acidity, 20, or .073% by wt.
Free HCl, absent.
Comb'd HCl, 14, or .051% by wt.
Tot. HCl, 14, or .051% by wt.
Acid salts, ——.

Lactic acid (direct), trace.
Starch, absent.
Amylodextrin, trace.

Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, trace.
Bile, absent.
Mucus, moderate amount.
Blood, trace.
Chymosin, deficient.
Peptonizing enzymes, absent.
Peptonizing zymogens, deficient.

At 2 a.m., the stomach contents were withdrawn from subject "S." The amount of chyme was ℥iv. Analysis showed: total acidity, .21% by wt.; free HCl, .11; combined HCl, .08. The K I absorption test showed the presence of iodin in the saliva in 16 minutes. The salol test was negative at all times.

Analysis of specimen taken at 2 a.m., July 24th, from subject "S," on the steamship *Southwestern*, after hypodermic injection of strychnin nitrate, gr 1/40, and atropin sulphate, gr 1/75. Ingested at 12.34 a.m. an Ewald test meal. Withdrawn at 2 a.m. Amount of chyme, ℥iv. Resorcin test showed the presence of free HCl.

Tot. acidity, 58, or .211% by wt.
Free HCl, 32, or .11% by wt.
Comb'd HCl, 24, or .08% by wt.

Tot. HCl, 56, or .19% by wt.
Acid salts, 2, or .007% by wt.

Lactic acid (direct), trace.
Starch, absent.

Amylodextrin, absent.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, present.
Coagulating enzymes, normal.
Peptonizing enzymes, normal.
Bile, absent.
Mucus, moderate amount.
Blood, absent.

The circulation was well maintained, as shown in protocol 19. The vaso-motor apparatus was evidently in a keen, steady state of activity and did its part in maintaining the blood-pressure without increase of the pulse-rate. Muscular activity was in part responsible for the high blood-pressure.

From the foregoing experience there can be no question of the beneficial effects of strychnin combined with atropin. The conditions of this journey were such that without these drugs both subjects would have been very seasick. Each subject after irrigation of the stomach went to bed and slept well. Both had a free action of the bowels on arising, and both felt well in every way.

July 24th.—At 11.22 p.m., the steamship *Southwestern* was boarded at Southampton, bound for Cherbourg. At 11.23 p.m., subject "S" received gr lx of potassium bromid, and at 11.50 subject "B" received, hypodermically, atropin sulphate, gr 1/80.

July 25th.—At 12.08 a.m., the boat started. At 12.36 subject "B" was given an Ewald test-meal. At 12.40 subject "S" was given a similar meal. The weather was not rough during this part of the night, but the boat rolled and pitched considerably. Both subjects experienced the "lump-sensation" in the stomach for a time. Neither subject at any time experienced more than a passing "feeling in the head," i.e., dizziness when the boat gave an unusual lurch. In subject "B" the sense of smell was slightly perverted, tobacco-smoke smelling like burnt rags.

The test was a fair one in so far as subject "B" was concerned, for owing to his sensitiveness to seasickness, the trip would undoubtedly have sufficed to sicken him thoroughly. Subject "S," however, is less susceptible, and would probably have been fairly well through such weather as was encountered. In subject "B" the atropin again caused a peculiar sour taste in the mouth. At 1.40 a.m., the test meal was withdrawn from subject "B's" stomach. The amount of chyme obtained was ʒiiss. When its contents had been evacuated, the stomach was irrigated and a good deal of mucus with a little blood was removed. Analysis showed: total acidity, .073% by wt.; free HCl, absent; combined HCl, .058. The K I absorption test showed a positive reaction in 14 minutes. The salol test did not react at any time.

Analysis of specimen taken July 25th, on steamship *Southwestern* from subject "B," after atropin sulphate, gr 1/80 hypodermically. Ingested at 12.36 a.m. an Ewald test-meal. Withdrawn at 1.40 a.m. Amount of chyme, ℥iiss. Reaction (litmus), acid. Free HCl absent (resorcin).

Tot. acidity, 20, or .073% by wt.
Free HCl, absent.
Comb'd HCl, 16, or .058% by wt.
Tot. HCl, 16, or .058% by wt.
Acid salts ——.
Lactic acid (direct), trace.

Starch, absent.
Amylodextrin, absent.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Bile, absent.
Mucus, considerable amount.
Blood, trace.
Enzymes, diminished.

At 1.50 a.m., the contents of subject "S's" stomach were withdrawn. The amount of chyme recovered was ℥v. Analysis showed: total acidity, .277% by wt.; free HCl, .189; combined HCl, .08. The K I absorption test showed a positive raection in 10 minutes. The salol test showed no reaction at any time. After evacuation of its contents, the stomach was irrigated.

Analysis of specimen taken July 25th on steamship *Southwestern* from subject "S" after KBr. gr lx. Ingested at 12.40 a.m. an Ewald meal. Withdrawn at 1.50 a.m. Amount of chyme, ℥v. Resorcin test showed the presence of free HCl.

Tot. acidity, 76, or .277% by wt.
Free HCl, 52, or .189% by wt.
Comb'd HCl, 22, or .080% by wt.
Tot. HCl, 74, or .269% by wt.
Acid salts, 2, or .007% by wt.
Lactic acid (direct), absent.
Starch, absent.

Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, present.
Bile, absent.
Mucus, moderate amount.
Blood, absent.
Enzymes, normal.

Whilst the test-meals were in the stomach, subject "B" felt somewhat drowsy, but subject "S" was wakeful. When the work with the test meals was finished, the weather became rather rough, but both subjects went to bed and slept soundly without suffering any ill effects. Cherbourg was reached about 7 a.m.

There can be little question that on this occasion atropin served subject "B" well in warding off the disagreeable effects which would certainly have been experienced without its use. It is to be noted here, as in rotations and aural irrigations, that atropin is of no benefit whatever in tending to relax the pylorus in the presence of irritation of the medullary centres. The bromid of potassium had an effect similar to that exhibited by it in rotations. It lessened psychic distress, and the fear of what was to come. On this occasion, however, it must be said that the conditions were not quite such as would properly test the efficiency of the drug.

July 26th.—At 9.50 p.m., the steamship *Southwestern*, bound for Southampton from Cherbourg, was boarded. At 9.55 p.m., subject "B" was given, *per os*, gr lx of potassium bromid. At 10.39 subject "S" received atropin sulphate, gr 1/50, *per os.*

July 27th.—At 12.06 a.m., each subject was given an Ewald meal.

At 12.17 subject "B" was given orthoform, gr v.

On this occasion the weather was fairly rough. The boat pitched and rolled considerably. Subject "B" experienced all the symptoms of the severe form of seasickness. The "lump-sensation" was present in the stomach and throat. There were also present nausea, increased salivary flow, pain in the left region of the epigastrium, nervousness and irritability of temper; general muscular weakness with tremulousness; a full, sickening feeling about the ears; occipital headache, worse toward the right side, a sense of fulness and lightness in the head, especially in the mastoid regions and behind the eyeballs. At times there was pain in the right as well as in the left epigastric region. The subject also felt chilly and drowsy. The face was flushed, the pupils normal, the conjunctivæ congested, and the skin cold. He was very anxious to lie down, which indeed he had to do in order to retain the test meal for the customary hour. At 1.07 a.m., the stomach contents of subject "B" were withdrawn. The amount of chyme recovered was ℥vi. Analysis showed: total acidity, .080 by wt.; free HCl, absent, and combined HCl, .058. The K I absorption test showed a positive reaction in 19 minutes. The salol test did not react at any time, the stomach having been irrigated after its contents had been withdrawn. The irrigation of the stomach gave the subject great relief from his symptoms, nevertheless each roll of the ship caused that indescribable sickening feeling which elsewhere has been called *dolor cerebri*. The subject was very glad to get back to bed.

Analysis of specimen taken July 27th on the steamship *Southwestern*, from subject "B" after receiving, *per os*, K Br., gr lx, and orthoform, gr v. Ingested at 12.06 a.m. an Ewald meal. Withdrawn at 1.97 a.m. Amount of chyme, ℥vi. Reaction (litmus), acid. Free HCl, absent (resorcin).

Tot. acidity, 22, or .080% by wt.
Free HCl, absent.
Comb'd HCl, 16, or .058% by wt.
Tot. HCl, 16, or .058% by wt.
Acid salts, ——.
Lactic acid (direct), trace.
Starch, absent.
Amylodextrin, absent.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, trace.
Bile, absent.
Mucus, moderate amount.
Blood, absent.
Enzymes, deficient or absent.

With the exception of a sense of fulness in the head, and a slight pain in the right, and also at times in the left epigastric region, subject "S" felt fairly comfortable.

At 1.20 a.m. his stomach contents were withdrawn. The amount of chyme recovered was ℥xii. The stomach was irrigated, and the symptoms previously mentioned disappeared, i.e., the pain in the epigastrium and the sensation of fulness in the head.

Analysis showed: total acidity, .255% by wt.; free HCl, .133; combined HCl, .109.

The K I absorption test showed a positive reaction in 24 minutes. The salol test did not react at any time, the stomach having been irrigated after its contents had been removed. The subject stood up all through the period during which the test meal was retained.

Analysis of specimen taken July 27th on steamship *Southwestern*, from subject "S," after atropin, gr 1/50, *per os*. Ingested at 12.06 a.m. an Ewald test meal. Withdrawn at 1.20 a.m. Amount of chyme, ℥xii.

Tot. acidity, 70, or .255% by wt.
Free HCl, 34, or .133% by wt.
Comb'd HCl, 30, or .109% by wt.
Tot. HCl, 64, or .242% by wt.
Acid salts, 6, or .021% by wt.
Lactic acid (direct), absent.
Starch, absent.
Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, present.
Bile, absent.
Mucus, moderate amount.
Blood, absent.
Enzymes, normal.

The experiences of this trip show that neither bromid of potassium nor orthoform has any potent action in counteracting the effects of seasickness. They also show that atropin *per os* may be used in seasickness to advantage, but not with such good effect as when used hypodermatically and in combination with strychnin. It was observed that the effect upon the bowels was not so happy after bromid as after atropin and strychnin.

The subject experienced a sour taste after the bromid, similar to that experienced with atropin.

July 27th.—A trip was made on the steamship *Lorna Doone* from Southampton to Southsea. The boat started at 9.15 a.m. The morning was fine and the water calm.

No ill effects were experienced, but on the return trip in the evening the weather was wet and dismal, and the boat rolled and pitched somewhat. Subject "B" felt many of the milder symptoms of seasickness, but the journey was too short to cause much trouble under the conditions prevailing.

CHAPTER XXVII

STUDIES IN SEASICKNESS (CONTINUED)

July 28th.—After a good night's rest in a cool room on shore at 7.18 a.m. each subject was given an Ewald test breakfast. At 8.25 the stomach contents of subject "B" were withdrawn. The amount of chyme recovered was ℥ii. It was mixed with much mucus. Free HCl was absent. Unfortunately the specimen was thrown away by accident before the quantitative examination was made. The K I test showed a positive reaction in 21 minutes. The salol test did not react at any time, the stomach having been irrigated when its contents had been removed.

At 8.30 the stomach contents of subject "S" were removed. The amount of chyme recovered was ℥ss. This specimen was also thrown away by mistake, but not until it had been ascertained that it contained free HCl. The amount of mucus was very moderate. The K I test showed a positive reaction in 16 minutes. The salol test reacted strongly in 1 hour 12 minutes. The condition of subject "B's" circulation after a restful night in a cool room is shown in protocol 19. The cardiac and neuro-vascular mechanisms were in good working order. The subject felt well in every way, except for an occasional feeling of indigestion.

At 11.52 a.m., the steamship *Teutonic*, bound for New York from Southampton, was boarded. As shown in protocol 20, the subject's circulation, before the boat started, manifested some depression incidental to the fatigue of making preparations to go on board. At 12.15 p.m. the boat started. The weather was mild and clear. The boat was very steady, but some slight vibration was felt from the machinery. Subject "B" felt well in every way. He took his meals and enjoyed them. After the evening meal, however, he had a slight feeling of indigestion. The circulation as shown in protocol 20 was efficiently maintained.

July 29th.—After lying all the morning in Queenstown Harbour, the pilot was put off at 1.15 p.m., and the boat got away under full steam. During the afternoon the boat pitched somewhat. This, with the jolting of the machinery, caused subject "B" to have occipital headache, worse upon the right side, with an occasional sick feeling in the head, as the ship made an unusually bad lurch. Toward 9 o'clock the ship was pitching considerably and subject "B" had the "lump-sensation" in the stomach to a marked degree. This was temporarily relieved by 10 drops of dilute HCl, in water, but it returned later and was as bad as ever. The effects of the pitching of the ship began to tell upon the circulation as seen in protocol 21. The vaso-motor tone was not as efficient in the late afternoon and evening as it had been earlier in the day.

July 30th.—At 7.40 a.m. both subjects felt very well after a good night. Each was given an Ewald breakfast. At 8.47 the stomach contents of subject "B" were evacuated. The amount of chyme recovered was ℥iii. Analysis showed absence of free HCl. Quantitative determinations were not made, owing to an accident. A considerable amount of mucus was present. The K I absorption test showed a positive reaction in 31 minutes. The salol test reacted in 55 minutes. At 8.59 the stomach contents of subject "S" were removed. The amount of chyme recovered was ℥iii. Analysis showed: total acidity, .262% by wt., free HCl, .175, combined HCl, .080. The K I test showed a positive reaction in 32 minutes. The salol test did not react at any time, the stomach having been irrigated after its contents had been removed.

Analysis of specimen taken July 30th on the steamship *Teutonic*, from subject "S." Ingested at 7.40 a.m. an Ewald breakfast. Removed at 8.59. Amount of chyme, ℥iii. Resorcin test showed the presence of free HCl.

Tot. acidity, 72, or .262% by wt.
Free HCl, 48, or .175% by wt.
Comb'd HCl, 22, or .080% by wt.
Tot. HCl, 70, or .255% by wt.
Acid salts, 2, or .007% by wt.
Lactic acid (direct), absent.
Starch, absent.
Amylodextrin, trace.
Erythodextrin, present.
Achroödextrin, present.
Maltose, present.
Peptones, present.
Bile, absent.
Mucus, moderate amount.
Blood, absent.
Enzymes, normal.

In the afternoon the sea became very rough and many people were made seasick. Subject "B" experienced no disagreeable symptoms except a "slight lump-sensation" in the stomach. At 5.11 p.m. the stomach-tube was introduced and ℥iss. of chyme removed. This was the remains of a meal taken at 1.10 p.m., and consisting of pea-soup, boiled codfish, mashed potatoes, boiled rice and water, ℥viii. Analysis showed free HCl, .073%. This subject "B" felt exceedingly well in spite of the very rough weather. Whatever slight trouble he experienced should be attributed, perhaps, to his indigestion. The condition of the circulation as represented in protocol 22 shows the vaso-motor mechanism to be thoroughly effective toward the latter part of the day.

July 31st.—On this day the weather was fair and the boat steady. After breakfast at 9.05 a.m. the K I absorption test showed a positive reaction in 1 hour and 5 minutes in subject "B." The salol motility test reacted in 2 hours and 22 minutes. This subject "B" felt very well all day. At times during the day he had slight intimations of indigestion. The condition of the circulation is shown in protocol 23. The blood-pressure was well maintained in the normal manner, excepting at times when the effects of the hot stuffy cabin manifested themselves by impaired vaso-motor tonus.

August 1st.—On this day the weather was warm, but on account of rain and mist the port-holes were kept closed. Subject "B" felt well in every way except for an occasional feeling of indigestion. He had no symptoms that could be attributed to the motion of the boat. The circulation as shown in protocol 24 was not properly maintained in the normal manner. There is evidence of vaso-motor inefficiency which is perhaps to be attributed to the effects of the warm stuffy cabin. The effects of hot and cold air upon the circulation are of importance not only in seasickness, but in any condition where the vaso-motor tone needs watching. As we have seen, time and again, seasickness is not due primarily to disturbance of the circulation and yet the latter is such a usual and constant phenomenon in the malady that whatever hinders or helps the efficiency of the circulatory mechanisms becomes a matter of moment to the sufferer. The good effect of cool fresh air upon the circulation is well shown in protocol 24, at 4.04 p.m., where there is every indication of keen vaso-motor efficiency. The difference between the torpid neuro-vascular mechanism and the neuro-vascular mechanism that is exhausted from overstimulation should constantly be remembered. In the former condition the mechanism is intact and measures to rouse it to activity may be employed, such as mild exercise, friction, thermic stimulation by cold bathing, and even drugs. In the latter condition, which is the characteristic one of true seasickness, the neuro-vascular mechanism is exhausted from overwork. For all practical purposes of therapy intended to directly enhance vaso-motor efficiency, there is no neuro-vascular mechanism, and it is irrational and harmful to try to stimulate the medullary centres or the sympathetic nervous system under these conditions. Here the problem has to be approached from the other side. If nothing may be done directly to enhance lessened vaso-motor efficiency the most should be made of what remains by conserving the circulatory mechanisms and diminishing to the utmost the demands put upon them. The principle of adaptation of the functional activities to the capacity of any organ or set of organs, or to the capacity of the organism as a whole, finds application in seasickness as in every other condition that faces the medical strategist. Any kind of treatment that offers hope must not be employed. A thorough understanding of the underlying causes and conditions is imperative in order that the best method of treatment in the individual case may be discovered and applied.

August 2d.—The effects of a hot, stuffy cabin upon the circulation are shown in parts of protocols 24 and 25. The contrast between the condition of the circulation after a night in a close cabin and when the port-holes were open later in the morning, challenges attention. See protocol 25. The vaso-motor efficiency from 11.36 a.m. until 9.53 p.m. was perfect. During all this time the port-holes were open, the temperature of the cabin being about 68° F.

After breakfast at 9.36 a.m. the K I absorption test showed in subject "B" a positive reaction in 27 minutes. At 9.55 a.m. subject "B" took ten drops of dilute HCl, to ascertain whether it would hasten the progress of food through the pylorus. It was noted that the salol test showed a positive reaction in exactly the same time that it took upon July 31st, viz., 2 hours and 23 minutes. Observations on subject "S" showed that the cool cabin had little effect in restoring vaso-motor efficiency. This subject's vaso-motor mechanism, as we have before remarked, is of an altogether different type from that of subject "B." Subject "S" seldom perspires freely. Cold bathing, if continued for any length of time, makes him blue all over and sets up actual rigours which last for some time. With subject "B" cold bathing causes the usual reaction, i.e., tonic hyperæmia with the glowing skin and general sense of well-being that accompanies it. Here is another indication of the necessity of closely studying the circulatory mechanism in every individual before recommending cold procedures in hydrotherapy.

August 3d.—In the early part of the day the weather was somewhat warmer, the temperature in the cabin being 74° F. at 7.47 a.m. The port-holes were open, but the cabin was on the leeward side and there was no fresh breeze blowing in. The effect upon the circulation is shown in protocol 26. The effects of the cool night are well shown. Subject "B" felt very well all day.

August 4th.—On this day the weather was cool, but wet and windy. There was little motion to the boat. The port-holes were closed all night on account of rain. The effect upon the circulation is shown in protocol 26. The subject felt well all day.

August 5th.—On awakening the ship was lying in New York Harbour. Subject "B" felt well in every way. The temperature in the cabin was 71° F., but the air was "stuffy." The effect upon the circulation is shown in protocol 26.

At 8 a.m. the passengers were landed in New York.

CHAPTER XXVIII

STUDIES IN SEASICKNESS (CONTINUED)

So far the experiments had shown that atropin in combination with strychnin is very effective in overcoming the chief disagreeable symptoms of seasickness. It was, however, apparent, notwithstanding the subjective sense of well-being, that the gastric functions were anything but normal. An effort was therefore made to find some means whereby the good effects of atropin and strychnin might be supplemented, the special object in view being improvement in gastric secretion, absorption and motility. A series of experiments was undertaken in which decoctions of the mucous membrane of the duodenum, and of the pylorus and other regions of the stomach, made after the manner described by Bayliss and Starling[91] and by Edkins,[92] were thoroughly tested by means of rotations. These experiments belong to a separate series and are detailed elsewhere. The results, however, warranted a trial of the decoctions in seasickness. Accordingly several sea-trips were undertaken with this object in view. Each of the three subjects used for the purpose of this study had been a long time under observation. Numerous test meals had been given to each on various occasions, so that the condition of the stomach in each as regards secretion, absorption and motility was thoroughly understood. During these tests observations of the pulse-rate and blood-pressure were also made as well as a study of the changes occurring in the retinal vessels during the milder forms of seasickness.

On November 8, 1910, a trip lasting about nine hours was made upon the steamship *Angler*. The weather was fair, but on the outward leg of the trip the boat pitched and rolled just enough to excite in all three subjects some of the milder symptoms of seasickness. During a period when the boat was at anchor the rolling was so bad that one of the subjects, "B," became very sick and succeeded in retaining his test meal for the accustomed hour only by remaining absolutely quiet and exercising all the self-control at his command.

Subject "C" had an instillation of homatropin in the right eye and the retinal vessels were studied with the ophthalmoscope at the commencement of, and several times during, the trip. As the boat started at 8.20 a.m. the retinal veins and arteries were somewhat constricted, the veins being slightly larger than the arteries. The blood-pressure at this time was 145 and the pulse-rate 92. At 8.52 the retinal vessels appeared to be slightly smaller, the white line of the arteries being more marked, although the retina as a whole was not very pale. The pupil was well dilated and yet some difficulty was experienced in studying the fundus at this period, because of the boat's motion and the unsteadiness of the candle-flame, an added factor being perhaps some slight changes in the refractive mechanisms. The blood-pressure at this time was 120 and the pulse-rate 76. At 11.52 the retinal veins were dilated as compared with the arteries which were very small. The retina looked paler than previously. The subject felt "a little queer in the head," i.e., heavy and groggy. At 11.21 the blood-pressure was 135 and the pulse-rate 84. This subject "C" has, in the receptors or afferent arcs related to his horizontal semicircular canals a certain degree of insensitiveness to aural irrigations, rotations and galvanism. This was manifested on numerous occasions in experimental tests. It was therefore a matter of interest to find that, on this trip as he sat with the coronal plane parallel to the long axis of the boat, the rolling motion (through his superior and posterior canals) so weakened him during the period that the boat lay at anchor that he could scarcely stand. This weakness was accompanied by nausea, vertigo and nystagmus, the latter being vertical toward the feet and accompanied by an apparent movement of external objects "away from him in front." At 8.32 an Ewald meal was given upon the fasting stomach and removed at 9.32. The chyme returned was ℥iv and contained a little fresh blood. At 10.16 a decoction of pig's pyloric mucous membrane, representing ⅓ of the yield of four stomachs, was given in its purity *per os* and followed by a few sips of water. At 10.25 an Ewald meal was given in which allowance was made for the fluid taken as pyloric decoction and as the mouth rinser that followed it. At 11.28 the stomach contents were withdrawn, the amount of chyme being ℥iv¼. With this meal the K I absorption test reacted for iodin in the mouth at 10.44, i.e., in nineteen minutes. The salol test showed no reaction within the hour and only a feeble reaction at 11.43.

Analysis showed for the first meal: total acidity, .343% by wt., free HCl, .211, combined HCl, .102; and for the second meal: total acidity, .284, free HCl, .226, combined HCl, .036.

The amount of free HCl after these two meals was unusual for the subject, who under somewhat similar circumstances, i.e., in rotations generally showed free HCl diminished or absent. As the subject had been up early and had had no food before 8.32 a.m., it is presumed that the resting state of the gastric glands coupled with normal hunger had something to do with the production of so much free HCl in the first meal, whilst in the second meal the high percentage of free HCl might perhaps be attributed in part to the pyloric decoction.

The latter assumption, however, gains no support from the results of the test meals given to the other subjects "B and S" in whom there was no distinct increase of free HCl in the test meals following the administration of pyloric extract. It is possible that in this test some of the gastric contents from the previous meal remained in the stomach, as there is no mention in the notes of the stomach having been irrigated after removal of the first Ewald meal.

Analysis of specimen taken Nov. 8th on the *Angler* from subject "C." Ingested at 8.30 a.m. an Ewald breakfast. Removed at 9.32. Amount of chyme, ℥iv. Resorcin test showed the presence of free HCl.

Tot. acidity, 94, or .343% by wt.
Free HCl, 58, or .211% by wt.
Comb'd HCl, 28, or .102% by wt.

Tot. HCl, 86, or .313% by wt.
Acid salts, 8, or .029% by wt.

Lactic acid, trace.
Starch, absent.

Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, trace.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, small amount.
Blood, present.

Note. A small amount of fresh blood came with the last of the stomach contents.

Analysis of specimen taken Nov. 8th on board the *Angler* from subject "C." Ingested at 10.25 a.m. an Ewald test meal following the administration *per os* of pyloric extract at 10.16. Withdrawn at 11.28. Amount of chyme, ℥iv. The chyme contained a few old blood-stained shreds but no free blood. Resorcin test showed the presence of free HCl.

Tot. acidity, 78, or .284% by wt.
Free HCl, 62, or .226% by wt.
Comb'd HCl, 10, or .036% by wt.
Tot. HCl, 72, or .262% by wt.
Acid salts, 6, or .021% by wt.

Lactic acid, trace.
Starch, absent.
Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, trace.
Peptones, present.
Bile, absent.
Mucus, very little.
Blood, absent.
Coagulating enzymes, normal.
Peptonizing enzymes (pepsin), subnormal.
Peptonizing zymogens (pepsinogen), subnormal.

Note. The test for pepsin and pepsinogen showed a poor degree of digestion as compared with the controls.

The tests made with subject "B" on board the *Angler* November 8th were as follows: The subject arose at 5 a.m. and had breakfast at 5.30, consisting of 6 oz. of bread with butter, 2 boiled eggs with butter 4 oz., and two cups of weak tea with milk and sugar. At 7.56 whilst the boat was still at the wharf the blood-pressure was 140 and the pulse-rate 96. This condition of the circulation was due to the heavy choppy motion of the boat, which caused marked dizziness in this susceptible subject. A few moments later the subject was sweating and the face and ears were flushed. At this time the blood-pressure and pulse-rate were 90 and 96 respectively. The cabin was warm and stuffy and the odour of bilge water extremely offensive. At 8.20 the boat started. During the first part of the journey the boat was fairly steady, but the air in the cabin was bad and affected the subject considerably. The vibration of the machinery was also annoying. The subject had a slight occipital ache with a sense of fulness in the head. The face was flushed and hot. The sight of the water rushing past the boat caused distress. There was nausea accompanied by chilly sensations along the spine. The subject sat with the coronal plane parallel to the long axis of the boat. At 9.08 the blood-pressure was 140 and the pulse-rate 96. The subject at this time was sitting in his shirt-sleeves in the path of a cool breeze from an open door. He felt weak and nervous all over. There was some nausea coupled with a slight "lump-sensation" in the stomach and a sickening heavy ache all through the head (*dolor cerebri*). Light was annoying to the eyes whilst noise of any sort was distinctly unpleasant. Psychic depression was marked. The boat rolled constantly, the movements being short and abrupt. At 10.01 the blood-pressure was 140 and the pulse-rate 84. At 10.50, after removal of its contents, the stomach was irrigated. The chyme amounted to ℥ iii and contained much mucus and a few old blood-stained shreds. At this time (11.07) the boat, lying at anchor, rolled very much. At 11.11 pyloric extract equivalent to ⅓ of the yield of four stomachs was given *per os*. This was followed by an Ewald test meal at 11.16 as in the case of subject "C." At 11.22 the subject felt heavy and wretched, the head ached, the face was flushed and warm, and the feet were cold. At 11.35 the blood-pressure was 140 and the pulse-rate 76. At 11.42 the subject felt a slight "lump-sensation" in the stomach toward the left of the epigastrium and with this a "groggy, drunken heaviness" in the head. The boat was still at anchor but rocking in a distressing manner. At 11.53 the subject felt nauseated and sick. The lump-sensation was present in the stomach, and the face was flushed. A moment later the subject was on the verge of vomiting; the skin became pale and broke out in sweat. At 12.05 the blood-pressure was 145 and the pulse-rate 76. The subject was still sweating, and very sick. Long deep breaths were frequently taken, the subject's weakness was extreme and he experienced a sense of suffocation as if "smoke were in the lungs preventing the entrance of the air." There were signs of marked irritability of temper which was, however, to a great extent controlled. After a few minutes the attack passed off. The subject's face assumed a better color; the feet became warm, but the muscles were still very tremulous and weak. The respirations just after the attack were from twelve to fourteen to the minute.

It will be noted that all through the above attack there was marked physical and psychic depression and yet the blood-pressure was high for this subject, i.e., 145 as compared with the normal, which for him is about 110. The pulse-rate was also remarkable in being rather slow, 76 as compared with the normal which averages for this subject 88 to 92. More remarkable still was the fact that at no time was there any gagging or contraction of the abdominal muscles. These observations show that lowered blood-pressure is not necessarily associated with the extreme sense of weakness that precedes vomiting. This fact, however, must not be interpreted as meaning that everything goes well with the circulation in the pre-vomiting stage. On the contrary; for whilst the peripheral vascular constriction with slowed heart-rate (both being the direct result of medullary irritation) may suffice to maintain a high blood-pressure yet the volume of the heart's output is so small in any given period that medullary anemia ensues. This latter condition may, to a great extent, be relieved by recumbency but nature has a most effective way of meeting it by vomiting which rids the subject of one of the powerful (secondary) causes of medullary irritation, viz., the gastric contents, and at the same time promotes the flow of abdominal blood toward the heart, i.e., raises the blood-pressure by mechanical means thereby obviating, to some extent, the necessity for extreme peripheral arterial constriction. Without entering into a discussion as to the mechanism of shock, i.e., as to whether shock is due to arterial relaxation (Crile) or to arterial constriction (Porter), or to venous relaxation (Henderson), it can be safely said that the end result of the vomiting is the same, viz., the supply of a greater volume of blood to the medullary centres with less effort on the part of the cardiovascular system.

At 12.18 the stomach contents were removed by vomiting. The amount of chyme recovered was ℥ixss. It contained a moderate amount of mucus, and a few old blood-stained scales. The salol, which had been administered in a gelatine capsule at the end of the meal, was vomited as a moist mass but intact, just as it had been swallowed, only minus the capsule. Owing to this fact in all subsequent experiments the salol was administered at the beginning of the test meals instead of at the end as in previous tests.

Immediately after vomiting, the subject felt very much better. At 12.37 the blood-pressure was 140 and the pulse-rate 76. The subject at this time was feeling fairly well but he had a slight headache and was very weak. As soon, however, as the boat weighed anchor and got under steam the cool air benefited him very much. At 6 p.m. the urine was normal. There was no evidence of albumin, sugar, or glycuronic acid.

Analysis of specimen taken Nov. 8th on board the *Angler* from subject "B." Ingested at 5.30 a.m.—breakfast consisting of 2 soft-boiled eggs with 4 oz. of butter; 2 cups of weak tea with milk and sugar and 6 oz. of bread with 2 oz. of butter. Removed at 10.50. The chyme was ʒ iii in amount and contained much thick mucus with a few tiny, old, blood-stained scales. Resorcin test showed absent or very greatly diminished free HCl.

Tot. acidity, 32, or .116% by wt.
Free HCl, 6, or .021% by wt.
Comb'd HCl, 20, or .073% by wt.
Tot. HCl, 26, or .094% by wt.
Acid salts, 6, or .021% by wt.
Lactic acid, present.
Starch, absent.
Amylodextrin, absent.
Erythrodextrin, absent.
Achroödextrin, absent.
Maltose, present.
Albumin, present.
Peptones, faint trace.
Coagulating enzymes (chymosin), present.
Peptonizing enzymes (pepsin), diminished or absent.
Peptonizing zymogen (pepsinogen), present.
Bile, absent.
Mucus, considerable amount.
Blood, absent.

Note. There was no evidence of gross fat in the chyme. Absorption and motility tests not made.

Analysis of specimen taken Nov. 8th on board the *Angler* from subject "B." Ingested at 11.16 a.m. an Ewald breakfast, preceded at 11.11 by pyloric extract given *per os* and representing ⅛ of the yield of 4 stomachs. Removed by vomiting at 12.16. The chyme amounted to ʒixss and contained a moderate amount of mucus with a few old, blood-stained scales. Resorcin test showed absent or considerably diminished free HCl.

Tot. acidity, 26, or .094% by wt.
Free HCl, 6, or .021% by wt.
Comb'd HCl, 14, or .051% by wt.
Tot. free HCl, 20, or .073% by wt.
Acid salts, 6, or .021% by wt.

Lactic acid, trace.
Starch, absent.
Amylodextrin, absent.
Erythrodextrin, trace.
Achroödextrin, present.
Maltose, present.
Albumin, present.
Peptones, faint trace.
Peptonizing enzymes (pepsin), diminished or absent.
Peptonizing zymogens (pepsinogen), present.
Coagulating enzymes (chymosin), present.
Bile, absent.
Mucus, moderate amount.
Blood, absent.

The K I absorption test showed iodin in the saliva in 17 minutes. The salol (motility) test did not react within the hour.

Experiments made November 8th on board the *Angler* upon subject "S." At 8.09 a.m. the blood-pressure was 135 and the pulse-rate 76. At this time the boat was beside the pier, and rocking very much. At 8.20 the boat started, and at 8.42 an Ewald meal was given upon the fasting stomach. At 8.54 the boat was fairly steady. The blood-pressure at this time was 140, and the pulse-rate 72. At 9.25 the blood-pressure was 130 and the pulse-rate 72. At 9.42 the stomach contents were removed. The chyme amounted to ℥iii¾, and contained nothing abnormal. During the journey so far, the boat had behaved fairly well. It pitched and rolled a little, however, just enough to cause disagreeable symptoms in subjects "B" and "C."

At 9.55 atropin sulphate, gr 1/50, was administered hypodermically. At 10.28 the blood-pressure was 130 and the pulse-rate 104. The rapid pulse, a very unusual thing in this subject, represents the atropin effect upon the vagus terminals in the heart. The boat was rolling somewhat at this period. At 10.33 pyloric extract, equivalent to ⅓ of the yield of 4 stomachs, was given *per os*. At 10.44 an Ewald test meal was given. At 11.30 the blood-pressure was 135, and the pulse-rate 92. At 11.42 the stomach contents were removed. The chyme amounted to ℥vss and contained nothing abnormal. During the time that the test meal was retained the subject complained of no disagreeable feelings although the boat rocked considerably as she lay at anchor. At 11.58 there was heaviness over the eyes. Otherwise the subject felt well. At 11.48 the blood-pressure was 125 and the pulse-rate 80.

Analysis of specimen taken Nov. 8th on board the *Angler* from subject "S." Ingested at 8.42 a.m. an Ewald breakfast. Removed at 9.42. The chyme was ℥ iii¼ in amount and contained nothing abnormal macroscopically. Resorcin test showed the presence of free HCl.

Tot. acidity, 80, or .292% by wt.
Free HCl, 54, or .197% by wt.
Comb'd HCl, 16, or .058% by wt.
Tot. HCl, 70, or .255% by wt.
Acid salts, 10, or .036% by wt.
Lactic acid, trace.
Starch, absent.
Amylodextrin, present.
Erythrodextrin, present.
Maltose, no marked reaction.
Albumin, faint trace.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, no excess.
Blood, absent.

Absorption and motility tests not made.

Analysis of specimen taken Nov. 8th on board the *Angler* from subject "S." Ingested at 10.44 a.m. an Ewald test meal, preceded by atropin sulphate, gr 1/50 hypodermically, at 9.55, and pyloric extract *per os* equivalent to ⅛ of the yield of 4 stomachs at 10.33. Removed at 11.44. The chyme amounted to ℥vss and contained nothing abnormal macroscopically. Resorcin test showed the presence of free HCl.

Tot. acidity, 54, or .197% by wt.
Free HCl, 26, or .094% by wt.
Comb'd HCl, 22, or .080% by wt.
Tot. HCl, 48, or .174% by wt.
Acid salts, 6, or .021% by wt.
Lactic acid, trace.
Starch, absent.
Amylodextrin, present.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, faint trace.
Peptones, present.
Peptonizing enzymes (pepsin), diminished.
Peptonizing zymogens (pepsinogen), diminished.
Coagulating enzymes, normal.
Bile, absent.
Mucus, no excess.
Blood, absent.

The K I absorption test showed iodin in the saliva in 19 minutes. The salol test showed no reaction within the hour. The pyloric extract used in the tests made on November 8th had been made some weeks and might therefore have undergone oxidation which seems to render it inert.

On November 20th another trip, lasting about eight hours, was made on the *Angler*. On this occasion tests were made upon the same subjects as in the previous experiments. As Edkins [92] has shown on animals that atropin does not affect the action of pyloric secretogogues, the action of this drug in conjunction with extracts of gastric mucous membrane was studied in subjects "B" and "C," whilst in subject "S" the effect of the extracts alone, i.e., without atropin was observed. The blood-pressure and pulse-rate and the general symptoms were also studied. In subject "C" the condition of the retinal vessels was studied.

Observations made November 20th on subject "C."

At 3.30 a.m. two glasses of diluted (half and half) milk were taken. At 6.30 one cup of water was taken and another at 7. The bowels moved rather freely at 6.30, 6.35 and 7. These movements were the result of cathartics taken on the previous day. At 8 a.m., after boarding the *Angler*, the blood-pressure was 140 and the pulse-rate 100. The boat started at 8.07. At 8.27 an instillation of homatropin was given in the right eye. At 8.42 the retinal arteries were about normal in size and of a slightly greyish color. The veins were somewhat larger than the arteries and of a dull red hue. The fundus as a whole was of a rich red colour. The boat was going steadily—no motion being manifested beyond the vibration from the engines. At 8.53 an Ewald meal was given. At 9.07 the blood-pressures were 140, 145, 140, 140, with corresponding pulse-rates of 100, 96, 100, 100. The boat was pitching and rolling somewhat at this period. At 9.35 the blood-pressure and pulse-rate were 135 and 100 respectively. At 9.40 the retinal vessels were about the same as when observed at 8.42. Just after the ophthalmoscopic examination of the eye the blood-pressure and pulse-rate were 135 and 96. At 9.53 the stomach contents were removed. The chyme was ℥iii¼ in amount and contained a moderate amount of mucus and a few old blood-stained scales. After removal of its contents the stomach was irrigated. At 10.25 atropin sulphate, gr 1/50, was given hypodermically. At this time the boat was rolling and pitching somewhat, and the subject, who was sitting with the coronal plane parallel to the long axis of the boat, felt a little distress in the head. At 10.47 the subject received, *per os*, extracts from the gastric mucous membrane of the pig (fundic and intermediate portions), each portion representing ⅓ of the yield of four stomachs. The bulk of the extracts was f℥v. At 11.04 the boat was rolling considerably. The blood-pressure and pulse-rate were 140 and 116 respectively. The pulse-rate evidently indicated the atropin effect upon the cardiac vagus terminals.

At 11.19 the boat, lying at anchor, rolled considerably and made the subject feel heavy and sleepy. At 11.20 the retinal arteries were distinctly smaller, whilst the veins were comparatively larger and darker. The fundus as a whole seemed paler than in previous observations. At 11.32 an Ewald test meal was given and removed at 12.32 p.m. The chyme was ℥iii in amount with no abnormal macroscopic contents. The K I absorption test showed iodin in the saliva in 15 minutes. The salol test showed a feeble, unreliable reaction at 12.30 but was distinctly positive at 1. From 12.39 to 12.42 the blood-pressures were 145, 145, 145, 145, with corresponding pulse-rates of 88, 88, 96, 92.

At 2 p.m. the retinal arteries were not so small as in the previous observations; the veins were a trifle larger than the arteries.

Analysis of specimen taken Nov. 20th on board the *Angler* from subject "C." Ingested at 8.53 a.m. an Ewald meal. Removed at 9.53. Amount of chyme, ℥iii¼. No abnormal macroscopic contents. Resorcin test showed the presence of free HCl

Tot. acidity, 70, or .255% by wt.
Free HCl, 50, or .182% by wt.
Comb'd HCl, 14, or .051% by wt.
Tot. HCl, 64, or .233% by wt.
Acid salts, 6, or .021% by wt.

Lactic acid, trace.
Starch, absent.

Amylodextrin, present.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, absent.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, no excess.
Blood, absent.

Note. The high percentage of free HCl in this meal, a very unusual thing for this subject, may be in part the result of the milk taken in the early morning, the products of digestion which lingered in the stomach acting as gastric secretogogues.

Analysis of specimen taken Nov. 20th on board the *Angler* from subject "C." Ingested at 11.32 a.m. an Ewald meal, after atropin sulphate, gr 1.50 hypodermically, at 10.25, and intermediate and fundic extracts representing for each portion ⅓ of the yield of 4 stomachs given *per os* at 10.47. Withdrawn at 12.32 p.m. Amount of chyme, ℥iii. No abnormal macroscopic contents. Resorcin test showed the presence of free HCl.

Tot. acidity, 54, or .197% by wt.
Free HCl, 40, or .146% by wt.
Comb'd HCl, 10, or .036% by wt.
Tot. HCl, 50, or .182% by wt.
Acid salts, 4, or .014% by wt.

Lactic acid, trace.
Starch, absent.

Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, absent.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, no excess.
Blood, absent.

Such a percentage of free HCl as shown in the above analysis never occurred in this subject before under similar circumstances, except in the tests of November 8th, when it resulted probably from secretogogues retained in the stomach from the previous meal. This, coupled with the fact that in the tests made upon subjects "B" and "S" there was unusual and marked increase of the free HCl, led to the supposition that the increased production of free HCl may have been the direct result of the gastric extracts.

The tests made on subject "S," November 20th, were as follows: The air was rather cold and sharp as the *Angler* was boarded. At 8.07 a.m. the boat started. The blood-pressure and pulse-rate at 8.10 were 115 and 80 respectively and at 8.13, 125 and 88. The boat had rocked rather freely at the pier but was fairly steady after the start. At 8.58 an Ewald breakfast was given. The weather was fine and everything looked merry in the bright sunlight. At 9.08 the blood-pressure and pulse-rate were 115 and 72 respectively. At 9.58 the stomach contents were removed and the stomach irrigated. The amount of chyme recovered was ℥ii with nothing abnormal macroscopically. At 10.44 the subject received, *per os*, extract of fundic and intermediate mucous membrane each portion representing ⅓ of the yield of four stomachs. The bulk of the extract was f℥v. At 11 the boat was rolling a little and the blood-pressure and pulse-rate were 110 and 72 respectively. At 11.19 an Ewald test meal was given. At this time the boat was lying at anchor and rolling with sickening effect. At 11.20 the stomach contents were removed. The chyme, which was ℥ iii in amount, contained nothing abnormal macroscopically. At 12.46 the blood-pressure and pulse-rate were 115 and 68 respectively. The subject experienced no marked disagreeable effects from the trip. After going home he had several loose movements from the bowels.

Analysis of specimen taken Nov. 20th on board the *Angler* from subject "S." Ingested at 8.58 a.m. an Ewald breakfast. Withdrawn at 9.58. Chyme amount ℥ ii. Nothing abnormal macroscopically. Resorcin test showed the presence of free HCl.

Tot. acidity, 88, or .321% by wt.
Free HCl, 64, or .233% by wt.
Comb'd HCl, 16, or .058% by wt.
Tot. HCl, 80, or .292% by wt.
Acid salts, 8, or .029% by wt.
Lactic acid, trace.
Starch, absent.

Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, trace.
Albumin, absent.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, no excess.
Blood, absent.

Absorption and motility tests not made.

Note. The high percentage of free HCl shown above, is unusual for this subject and may, perhaps, be explained by the resting state of the glands and the long fast. Ordinarily the subject eats breakfast at 5.30 a.m., whereas in this test the first food was taken at 8.58 a.m.

Analysis of specimen taken Nov. 20th on board the *Angler* from subject "S." Ingested at 11.19 a.m. an Ewald meal. The subject had received, *per os*, at 10.44 extract of fundic and intermediate mucous membrane, each part representing ¼ of the yield of 4 stomachs. Withdrawn at 12.20 p.m. Amount of chyme, ℥iii. Nothing abnormal macroscopically. Resorcin test showed the presence of free HCl.

Tot. acidity, 94, or .343% by wt.
Free HCl, 72, or .262% by wt.
Comb'd HCl, 20, or .073% by wt.
Tot. HCl, 92, or .335% by wt.
Acid salts, 2, or .007% by wt.
Lactic acid, trace.
Starch, absent.

Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, absent.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, no excess.
Blood, absent.

The K I absorption test showed the presence of iodin in the saliva in 15 minutes. The salol did not react within the hour

but reacted positively in 1 hour and 45 minutes. The high percentage of free HCl seems to have been due in part to the effect of the gastric extracts.

The tests made on subject "B" November 20th, on board the *Angler*, were as follows: The subject was detained unexpectedly and had to run 650 yards at a good pace, so as not to miss the boat. After going on board he experienced some tightness in the chest as if smoke were in the bronchial tubes, preventing the free ingress of air. At times he coughed like an asthmatic. Undoubtedly the subject's symptoms were due to spasm of the bronchial muscles. A moment after entering the cabin the subject began to perspire but the tightness in the chest still persisted. At this time the subject's anger was aroused by the obstinacy of a disagreeable individual who would not move his chair a couple of inches, so as to afford room for the blood-pressures to be taken with comfort to all concerned. Words were exchanged and for a moment it looked as though the members of the party might have to defend themselves against the disagreeable individual and his friends. Good sense and sober judgment prevailed however, which was a good thing for the instruments and perhaps for a certain burly bully. The blood-pressures which were observed all through the period of possible impending struggle, i.e., from 8.16 to 8.20 were 155, 150, 150, 140, 140, with corresponding pulse-rates of 104, 100, 104, 100, 100. At 8.30 the subject felt well and was perspiring slightly. The blood-pressure and pulse-rate were 130 and 100 respectively. At this period the subject felt a slight ache and sense of fulness in the head. There were also some eructations. The boat was rocking slightly, and the room was filled with tobacco-smoke. From 9.12 to 9.22 the blood-pressures were 135, 130, 125, 115, 120, 120, 115, 120, 115, and 115, with corresponding pulse-rates of 96, 96, 100, 100, 96, 96, 92, 92, 92, and 96.

At 9.34 the blood-pressure and pulse-rate were 115 and 96 respectively. At this period the subject had a slight headache, and his face was flushed and hot. Air blew into the room fresh and cool from an open door. At 8.48 the boat was rolling and pitching to some extent. The subject had a slight occipital headache on the right side, extending to the ear. The face was still flushed and warm. At 9.53 the skin was moist from perspiration, and the subject felt a little sick with a slight "lump-sensation" in the stomach. At 10.37 atropin sulphate, gr 1/50, was given hypodermically. At 10.55 the face was flushed, and the head felt heavy. At 11 the boat was still rolling somewhat. The subject experienced fulness in the head, and had a tendency to sleep. The face was still flushed. From 11.08 to 11.15 the blood-pressures were 120, 125, 125, 125, with corresponding pulse-rates of 88, 92, 92, 92. At this period the boat lay at anchor, and rolled with sickening effect. The subject's mouth was dry from the atropin. The rolling of the boat caused a little distress in the head. At 11.40 the stomach contents were removed. The chyme, which amounted to ℥ii¾, contained much mucus, a few old blood-stained scales and, toward the end, a very small amount of gross fat with a little fresh blood. After removal of its contents the stomach was irrigated.

At 11.57 the subject was given, *per os*, extract of fundic and intermediate mucous membrane of each an amount equivalent to ⅛ of the yield of 4 stomachs. At 12.05 p.m. there was a slight pain in the right epigastric region. At 12.23 flatus was passed, and there was pain in the region of the left iliac fossa. The subject felt well except for those pains low down in the abdomen. At 12.32, an Ewald test meal was given. The usual sour taste which so often follows the administration of atropin was experienced as the subject ate his test meal. At 12.38 there was a cool pleasant sensation in the stomach. There were no unpleasant symptoms of any kind, but the face was slightly flushed. At 12.43 there were intermittent sharp colicky pains in the lower abdomen. From 12.52 to 12.55 the blood-pressures were 125, 130, 130, 130, with corresponding pulse-rates of 76, 80, 80, 80. At 1.30 the stomach contents were removed. Owing to persistent gagging, it was unusually hard to introduce the stomach-tube. The chyme, which amounted to ℥iii, contained a moderate amount of mucus, and a few old blood-stained scales, but no free blood. At 1.50 the blood-pressures were 135, 135, 130, 135, with corresponding pulse-rates of 68, 72, 72, 72. At 2.05, the subject felt well and enjoyed a hearty dinner, which was followed by a large loose evacuation of the bowels. Evidently the atropin and perhaps the gastric extracts stimulated gastro-intestinal motility, for the other subjects were somewhat similarly affected, especially subject "S."

It is noteworthy that the atropin in the dose given had no effect in releasing the heart from vagus control in this subject as in the others. This fact has been repeatedly observed in subject "B" in whom atropin in fair dosage generally tends to slow the pulse-rate if anything.

Analysis of specimen taken Nov. 20th on board the *Angler* from subject "B." Ingested at 6 a.m. 2 soft-boiled eggs with 4 oz. of butter; 2 cups of weak tea, with milk and sugar, and 6 oz. of bread with 2 oz. of butter. Withdrawn at 11.40 a.m. Amount of chyme, ℥ii¼. Macroscopically there was a considerable amount of mucus with a few old blood-stained scales, a trace of free fat and a small amount of fresh blood. Resorcin test showed the absence of free HCl.

Tot. acidity, 44, or .160% by wt.
Free HCl, ——.
Comb'd HCl, 26, or .095% by wt.
Tot. HCl, 26, or .095% by wt.
Acid salts, ——.
Lactic, trace.
Starch, absent.
Amylodextrin, absent.
Erythrodextrin, absent.
Achroödextrin, absent.

Maltose, trace.
Albumin, present.
Peptones, present.
Peptonizing enzymes (pepsin), diminished or absent.
Peptonizing zymogens (pepsinogen), present.
Coagulating enzymes (chymosin), present.
Bile, absent.
Mucus, some excess.
Blood, present.

Note. The old blood-stained scales so often alluded to represent epithelium and exudate from the gastric mucous membrane. Evidently subject "B" suffered from a mild grade of chronic gastritis.

Analysis of specimen taken Nov. 20, 1910, on board the *Angler* from subject "B." Ingested at 12.32 p.m. an Ewald meal. Atropin sulphate, gr 1/50, had been given hypodermatically at 10.37 a.m. and gastric extract, fundic and intermediate, of each an amount equivalent to ⅛ of the yield of 4 stomachs at 11.57 *per os*. Withdrawn at 1.30 p.m. Amount of chyme, ℥iii. Macroscopically there was a moderate amount of mucus and a few old blood-stained scales. Resorcin test showed the presence of free HCl.

Tot. acidity, 58, or .211% by wt.
Free HCl, 36, or .131% by wt.

Comb'd HCl, 22, or .080% by wt.
Tot. HCl, 58, or .211% by wt.

Acid salts, none.
Lactic acid, absent.
Starch, absent.
Amylodextrin, absent.
Erythrodextrin, present.
Maltose, trace.
Albumin, absent.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, moderate amount.
Blood, absent.

The K I absorption test showed the presence of iodin in the saliva in 12 minutes. The salol test reacted positively in 1 hour and 3 minutes.

Note. The high percentage of free HCl shown in the above analysis is unique for this subject. In all analyses following Ewald test meals given this subject, even in the resting state upon shore, free HCl was very low or absent. It seems, therefore, justifiable to attribute, in part at least, the enhanced production of free HCl in this and the other cases of this day's tests to the effects of the gastric extracts.

On November 24th another trip lasting about eight hours was made on the *Angler*. The same subjects, viz., "B," "C," and "S," were used as in the preceding tests. The details of the tests made on subject "B" are as follows: At 5.50 a.m. breakfast was taken, consisting of several slices of toast representing about ʒvi of bread, with ʒii of butter and 2 cups of tea, to which were added milk and sugar. At 8.02 the blood-pressure was 135 and the pulse-rate 84. At this time the boat was still at the pier, but was rocking very much from the wash of passing craft. The subject had arisen early, and had plenty of time to reach the boat, so that there was no hurry or rush as on the previous day. When the blood-pressure above recorded was taken, he was sitting in his shirt-sleeves in a cool atmosphere. At 8.15 the boat started. The weather was fair and cool, but the skies were overcast. The boat rolled and pitched somewhat. At 8.30 the subject's face was flushed and hot, and there was a sense of pressure across the abdomen, about the level of the umbilicus. The cabin was filled with tobacco-smoke, the air being anything but fresh. At 8.40 the blood-pressures were 130, 130, 135, 135, with corresponding pulse-rates of 72, 76, 76, 76. At 8.48 the subject had slight headache with flushed face. At 8.59 there was still some slight headache, and a little nausea. The odour of tobacco-smoke was distinctly unpleasant. The gloom of the day seemed to attach itself to everything. The subject felt warm. At this period the boat was rolling considerably, and the sound of the engines reverberated through the head in a distressing manner. At 9.12 the blood-pressures were 125, 125, 125, 130 with corresponding pulse-rates of 76, 76, 80, 80. At this period there was dizziness and heaviness in the head with some headache, "lump-sensation" and a distinct tendency to sickness of the stomach. At 9.29 an Ewald test meal was given. The stomach had not been irrigated before this meal. The boat at this time was rolling considerably, and the subject felt some headache and *dolor cerebri*. At 9.34 the boat was still rolling somewhat, and the subject felt "sick in the head" with pain in the occipital region, and heaviness about the eyes. The sense of tension in the muscles of the abdomen persisted. At 9.38 the boat was rolling and pitching badly. At 10.14 the subject felt heavy, and there was a burning sensation in the stomach accompanied by eructations. At 10.30, the boat was still rolling and pitching. The subject felt heavy, and slightly sick. There was a distinct "lump-sensation" in the stomach. At 10.29 the gastric contents were removed and the stomach irrigated. The chyme amounted to ℥v and contained much mucus and a few old blood-stained scales. These latter were much less than after previous breakfasts in which eggs had been taken.

At 10.46 the boat lay at anchor, but it rolled constantly with sickening effect. At 11.02 atropin sulphate, gr 1/50 was given hypodermically. At this time the subject felt somewhat distressed from the constant rolling. At 11.12 the subject received, *per os*, pyloric extract representing ⅔ of the yield of four stomachs. The bulk of the extract was f℥iv. At 11.30 the blood-pressures were 125, 125, 130, 130, with corresponding pulse-rates of 76, 76, 72, 72. The boat was still rolling and the subject had a slight headache. The mouth was dry from the atropin. The odour of tobacco-smoke seemed somewhat changed, although not as unpleasant as usual. The stomach manifested no disagreeable symptoms. At 11.40 the mouth was very dry and the subject felt much better and stronger. At 11.53 an Ewald meal was given. No sour taste was experienced as on previous occasions after atropin. At 11.57 strychnin sulphate, gr 1/30, was given hypodermically. The subject felt well in every way, and experienced a nice cool sensation in the stomach. He noticed, however, that when he attempted to walk he was unable to balance very well, although the tossing about did not cause much dizziness or distress. Similar incoordination following the use of atropin at sea was frequently observed in "B" and other subjects. At 12.10 the subject still felt well in every way. There were some eructations. The knee-jerks were somewhat active. From 12.16 to 12.21 the blood-pressures were 130, 125, 130, 125, 120, 115, 120, with corresponding pulse-rates of 88, 88, 88, 92, 92, 88, 92. The boat was still rolling very much, but the subject felt well in every way. There were some eructations, however. At 12.48 the boat was still rolling very much just off Sandy Hook. The subject's nose and mouth were very dry, but otherwise he felt quite well. At 12.58 the stomach contents were removed. The amount of chyme recovered was ℥ss. It contained nothing abnormal macroscopically. At 1.10 the blood-pressures were 130, 130, 135, 130 with corresponding pulse-rates of 80, 84, 80, 80. At this time the boat was rolling considerably. The air was chilly, but the subject experienced no effect beyond dryness of the mouth and nose, and a slight, not unpleasant sense of lightness in the head.

Analysis of specimen taken Nov. 24th on board the *Angler* from subject "B." Ingested at 9.29 a.m. an Ewald test meal. The stomach had not been irrigated and the subject had taken breakfast at 5.50 a.m., consisting of toast, equivalent to ℥vi of bread with 2 oz. of butter and two cups of tea with milk and sugar. Removed at 10.29. Amount of chyme, ℥v. Macroscopically there was much tenacious mucus and a few old blood-stained scales. Resorcin test showed the presence of free HCl.

Tot. acidity, 42, or .153% by wt.
Free HCl, 24, or .087% by wt.
Comb'd HCl, 14, or .051% by wt.
Tot. HCl, 38, or .138% by wt.
Acid salts, 4, or .014% by wt.
Lactic acid, trace.
Starch, absent.

Amylodextrin, absent.
Erythrodextrin, trace.
Achroödextrin, present.
Maltose, present.
Albumin, trace.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, excessive amount.
Blood, absent.

Note. The products of digestion left over in the stomach from the breakfast at 5.50, were undoubtedly the cause of the fair gastric secretion represented in the analysis above. In similar test meals tried at various times upon the subject, on a fasting stomach or after irrigation the tests always showed diminished or absent free HCl. Absorption and motility tests not made.

Analysis of specimen taken Nov. 24th on board the *Angler* from subject "B." Ingested at 11.53 a.m. an Ewald meal. Atropin sulphate, gr 1/50, had been given hypodermically at 11.02 a.m. and pyloric extract ½ of the yield of 4 stomachs, *per os*, at 11.12. At 11.57 strychnin sulphate, gr 1/30, was given hypodermically. Withdrawn at 12.53 p.m. Amount of chyme, ℥ss. Nothing abnormal macroscopically. Resorcin test showed a very feeble reaction for free HCl.

Tot. acidity, 30, or .109% by wt.
Free HCl, 10, or .036% by wt.
Comb'd HCl, 10, or .036% by wt.
Tot. HCl, 20, or .072% by wt.
Acid salts, ——.

Lactic acid, absent.
Starch, absent.
Amylodextrin, absent.
Erythrodextrin, trace.

Achroödextrin, present.
Maltose, present.
Albumin, trace.
Peptones, absent.
Peptonizing enzymes (pepsin), absent.
Peptonizing zymogens (pepsinogen) present.
Coagulating enzymes, normal.
Bile, absent.
Mucus, moderate amount.
Blood, absent.

The K I absorption test showed the presence of iodin in the saliva in 11 minutes. The salol test showed a positive reaction in 1 hour and 42 minutes.

The low percentage of free HCl may, perhaps, have been due in part, to fatigue of the gastric glands following the previous meal. Absorption and motility were evidently favoured by the pyloric extract as well as by the atropin and strychnin. Owing to the small amount of chyme the quantitative analysis involved some difficulty, but the figures given are fairly reliable, especially when taken in conjunction with the result of the resorcin test.

The details of the tests made November 24th on subject "C" were as follows: At 7 a.m. the subject took a cup of sweetened lemonade. At 8 homatropin was instilled into the right eye. At 8.05 on board the *Angler*, which was still beside the pier, the blood-pressures were 145, 140, 140, with corresponding pulse-rates of 100, 104, 104. The boat was much disturbed by the wash from passing craft. At 8.15 the boat started. At 8.28 the subject drank ℥viii of water. At 8.38 an Ewald meal was given, but with only ℥ii of water, as the subject had had ℥viii of water at 8.28. At 8.44 the blood-pressures were 130, 130, 135, 140, 140, with corresponding pulse-rates of 96, 96, 96, 96, and 100. The slight rise in the last two pressures may have been due to a scolding given the subject for taking water without permission and for failing to report having taken it. At 8.58 the retinal arteries were moderately constricted, the veins being somewhat larger. At this time the subject felt a heaviness in the head, due to the rolling and pitching which had been considerable. At 9.16 the blood-pressures were 145, 140, 140, with corresponding pulse-rates of 100, 100, 96. At 9.38 the stomach contents were removed. The chyme amounted to ℥iii, and was of a dark brown (chocolate) colour, with about half an ounce of gross fat floating on the surface. This was the result of chocolate-vanilla ice-cream taken at 10 p.m., on the preceding evening. At 9.57 atropin sulphate, gr 1/50, was given hypodermically. At 9.58 the subject was feeling badly, and had a frontal headache. There was no "lump-sensation" present. At 10 the blood-pressures were 140, 140, 140, 140, with corresponding pulse-rates of 88, 88, 92, 92. At 10.15 the subject received, *per os*, extract of the fundic mucous membrane, equivalent to ⅓ of the yield of 4 stomachs and a similar amount of pyloric extract. The bulk of the extract, in a watery diluent, was f℥v. At 10.25 the retinal arteries were somewhat larger than they had been in the previous observation.

The veins were relatively somewhat smaller, but were still a trifle larger than the arteries. At 10.50 strychnin sulphate, gr 1/30, was given hypodermically. At 10.55 an Ewald meal was given. At 10.51 the knee-jerks were absent even with Jendrassik's reinforcement. At 11.05 the subject felt "bad in the head." There was frontal headache, as if a tight band were tied about the head. The stomach felt very well. There was no "lump-sensation" and no eructations. At this period there was no nystagmus. At 11.38 the blood-pressure was 145, and the pulse-rate 100. The knee-jerks were still absent, even with reinforcement. At 11.55 the stomach contents were removed. The chyme was ℥v in amount and was of a yellowish colour. There was no evidence of gross blood. At 12.10 p.m. the knee-jerks were still absent, even with reinforcement. At 12.23 the blood-pressures were 140, 145, 145, 145, with corresponding pulse-rates of 84, 92, 92, 92. The boat at this time was rolling considerably. At 1.18 the blood-pressures were 140, 145, 145, 145, with corresponding pulse-rates of 84, 84, 84, 88. At 1.30 there was a sour taste in the mouth, due probably to the atropin. About this time it was suggested that the party retire to the dining-room, but the subject had no desire for food. At 2.15 he ate a fairly large dinner, consisting of clam chowder, turkey with dressing, a cup of coffee, plum-pudding, and mince-pie. This dinner was taken, although the subject knew from previous experience, that it would sicken him. At 3.20 he was compelled to empty the stomach, which he did in his accustomed way by tickling the pharynx with his forefinger. At 3.30 the knee-jerks were not only present without reinforcement, but were somewhat exaggerated. From 3 it was evident that the subject was beginning to have one of the typical gastric crises of tabetic origin of which he was a victim.

Analysis of specimen taken Nov. 24th on board the *Angler* from subject "C." Ingested at 8.38 a.m. an Ewald meal. The subject had taken one cup of sweetened lemonade at 7 a.m. Withdrawn at 9.38. Amount of chyme, ℥iii. On macroscopic examination the chyme was found to be dark brown in colour with about half an ounce of free fat floating on the surface. Resorcin test showed the presence of free HCl.

Tot. acidity, 68, or .248% by wt.
Free HCl, 44, or .160% by wt.
Comb'd HCl, 14, or .051% by wt.
Tot. HCl, 58, or .211% by wt.
Acid salts, 10, or .036% by wt.

Lactic acid, present.
Starch, absent.

Amylodextrin, absent.
Erythrodextrin, trace.
Achroödextrin, present.
Maltose, present.
Albumin, absent.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, moderate amount.
Blood, absent.

Note. The stomach evidently contained normal gastric juice at the beginning of the meal, and the products of digestion acted as gastric secretogogues since the percentage of free HCl shown in the analysis is very unusual for this subject after similar test meals. The marked stasis of food taken on the previous evening is noteworthy as a precursor of the gastric crisis which was evidently hurried on by the atropin and strychnin and perhaps by the gastric extracts for which the subject has a positive dislike. The enhanced secretion of free HCl may have been due to the early mild irritation of the medullary centres incidental to the approach of a gastric crisis.

Analysis of specimen taken Nov. 24th on board the *Angler* from subject "C." Ingested at 10.55 a.m. an Ewald meal. Atropin sulphate, gr 1/50, had been given hypodermically at 9.57 and strychnin sulphate, gr 1/30, at 10.50, also hypodermically. The subject also had received, *per os*, at 10.15 fundic extract equivalent to ⅛ of the yield of 4 stomachs and a similar amount of pyloric extract. The bulk of the extracts with their watery diluent was ℥v. Withdrawn at 11.55. Amount of chyme, ℥vss. On macroscopic examination the chyme was of yellowish colour with no evidence of blood. Resorcin test showed a very feeble reaction for free HCl.

Tot. acidity, 26, or .095% by wt.
Free HCl, 10, or .036% by wt.
Comb'd HCl, 12, or .044% by wt.
Tot. HCl, 22, or .080% by wt.
Acid salts, 4, or .014% by wt.
Lactic acid, trace.
Starch, absent.
Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.

Maltose, present.
Albumin, trace.
Peptones, faint trace.
Peptonizing enzymes, absent.
Peptonizing zymogens, diminished.
Coagulating enzymes, absent.
Coagulating zymogens, present.
Bile, absent.
Mucus, moderate amount.
Blood, absent.

The K I absorption test reacted in 12 minutes. The salol motility test reacted in 1 hour and 5 minutes.

The inefficiency of fundic and pyloric extract to induce secretion of a normal gastric juice in spite of the fact that absorption and motility were enhanced is noteworthy. The absence of symptoms referable to the stomach is to be attributed to the atropin, although this latter drug is quite inefficient in combating the distress referred to the stomach in gastric crises. The absence of skipped beats in the pulse during the forenoon and their reappearance on the approach of the gastric crisis is also noteworthy. Whether the present test made on this particular subject is a fair one as to the efficiency of the combinations of pyloric and fundic extract is an open question. The failure of gastric secretion can hardly be attributed to pronounced medullary irritation from the oncoming gastric crisis, because in that case we might expect marked gastric stasis and failure of absorption from the same cause, whereas the gastric motility and absorption were distinctly enhanced after strychnin and atropin, etc. The failure of normal gastric secretion might possibly be attributed to fatigue of the gastric glands, but in a previous test where intermediate extract was used, no such fatigue effects were manifested. The fact that in subject "B" the gastric secretion was deficient, throws no light on the subject, since in his case only pyloric extract was used.

In subject "S" to whom intermediate extract was given the gastric juice secreted was normal or enhanced for this subject. It remains therefore a question as to whether the failure of gastric secretion in the case of "B" and "C" was due to fatigue or other cause, or to the inefficiency of fundic and pyloric extracts to stimulate the gastric glands to activity. Pending further investigation, the latter view seems the more probable.

The details of the tests made November 24th upon subject "S" are as follows: At 8.10 a.m. on board the *Angler* which, though still at the pier, was rocking very much in the wash of passing craft, the blood-pressures were 130, 135, 130, 130, with corresponding pulse-rates of 72, 68, 76, 68. At 8.15 the boat started and at 8.20 the fasting stomach was irrigated. At 8.29 an Ewald breakfast was given. The boat at this period was rolling and pitching somewhat. At 8.48 the blood-pressures were 125, 125, 125, 125, 125, with corresponding pulse-rates of 76, 76, 76, 68, 64. At 9.20 the blood-pressures were 125, 115, 115, 115, with corresponding pulse-rates of 68, 64, 68, 68. At 9.29 the stomach contents were removed. The chyme was ℥iii in amount, and was thin in consistency and of a yellowish colour. At 9.58 atropin sulphate, gr 1/50, was given hypodermically. At 10 the subject felt very well. At 10.05 the blood-pressures were 125, 125, 125, 125, with corresponding pulse-rates of 56, 60, 60, 56. The boat was still rolling a good deal. At 10.08 the subject received, *per os*, "intermediate extract" equivalent to ⅓ of the yield of 4 stomachs and a similar amount of fundic extract. The extracts with their watery diluent amounted to f℥v. At 10.45 an Ewald meal was given, and at 10.57 strychnin sulphate, gr 1/30, was given hypodermically. At 11.06 the subject felt a band sensation around the head. At 11.17 there was slight dizziness, although the stomach felt very well. There was no nystagmus visible to the naked eye. At 11.40 the blood-pressures were 125, 120, 120, 115 with corresponding pulse-rates of 80, 80, 84, 80, The increased pulse-rate was evidently the effect of the atropin on the cardiac vagus terminals. At 11.45 the stomach contents were removed. The chyme amounted to ℥iiss and was of a yellowish colour with little or no mucus. At 12.10 the knee-jerks were active, but resembled movements of voluntary extension rather than the sharp response of the normal knee-jerk. At 12.30 the blood-pressures were 105, 110, 110, with corresponding pulse-rates of 64, 68, 68. Evidently the effects of the atropin upon the cardiac vagus terminals and upon the vaso-motor mechanisms had worn off. At 1.20 the blood-pressures were 105, 105, 110, 105, 105 with corresponding pulse-rates of 68, 60, 64, 60, 60. At 1.24 the subject felt very well. At 1.30 there was no great desire to eat.

Analysis of specimen taken Nov. 24th on board the *Angler* from subject "S." Ingested at 8.29 a.m. an Ewald breakfast. The fasting stomach had been irrigated at 8.20 a.m. Removed at 9.29. Amount of chyme, ℥iii. Macroscopically the chyme was of yellowish colour, thin in consistency and contained no excess of mucus. Resorcin test showed the presence of free HCl.

Tot. acidity, 64, or .233% by wt.
Free HCl, 54, or .197% by wt.
Comb'd HCl, 4, or .014% by wt.
Tot. HCl, 58, or .211% by wt.
Acid salts, 6, or 021% by wt.

Lactic acid, absent.
Starch, absent.

Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, absent.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, no excess.
Blood, absent.

Absorption and motility tests not made.

Note. The free HCl content may here be fairly attributed to the activity of the well-rested gastric glands.

Analysis of specimen taken Nov. 24th on board the *Angler* from subject "S." Ingested at 10.45 a.m. an Ewald meal. At 9.53 the subject received hypodermically atropin sulphate, gr 1/50. At 10.08 he received, *per os*, fundic and intermediate gastric extract of each an amount equivalent to ⅓ of the yield of 4 stomachs. At 10.57 strychnin sulphate, gr 1/30, was given hypodermically. Removed at 11.45. Amount of chyme ℥iiss. Macroscopically the chyme was yellowish in colour and contained nothing abnormal. Resorcin test showed the presence of free HCl.

Tot. acidity, 56, or .204% by wt.
Free HCl, 40, or .146% by wt.
Comb'd HCl, 14, or .051% by wt.
Tot. HCl, 54, or .197% by wt.
Acid salts, 2, or .007% by wt.

Lactic acid, absent.
Starch, absent.

Amylodextrin, trace.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, absent.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, no excess.
Blood, absent.

The K I absorption test reacted in 18 minutes. The salol test reacted in 1 hour and 20 minutes. The fairly normal gastric juice of the above analysis may fairly be attributed in part, at least, to the effects of the gastric extracts, as the percentage of free HCl is higher than that obtained in similar test meals in the resting condition on shore. It was again observed that in subjects "B" and "S" the bowels moved rather freely, on the same afternoon in the case of "S," and on the next morning in the case of "B." The latter subject has a tendency to constipation. Subject "C's" bowels did not move that evening or the next day, but he received morphin sulphate, gr ss., hypodermically about 5 p.m. as he had developed one of his usual attacks of true gastric crises.

On January 8th the effect of hypnotic suggestion was studied in subject "F." At 7 a.m. the subject drank ℥iv of water. At 8.07, on board the *Angler*, the ear drums were normal, there being no blood-vessels visible. At 8.15 the blood-pressures were 125, 125, and 125 with corresponding pulse-rates of 92, 92, and 88. At 8.20 the boat started. On ophthalmoscopic examination under homatropin, the retinal arteries were moderately dilated the veins being somewhat larger. The fundus jerked toward the left about every three seconds, at times executing this movement toward the left in one sharp movement, and at other times in a series of small jerks. Later, the nystagmic movements of the fundus toward the left (of the patient) were almost constant. During these observations the subject sat erect, the coronal plane of his body being parallel to the long axis of the boat, and his right side directed toward the bow. At this time the boat was rolling moderately.

At 8.51 the blood-pressures were 130, 135, 135, and the corresponding pulse-rates 80, 84, and 84. At 9.09 an Ewald meal was given. At 9.12 the blood-pressures were 135 and 135, the pulse-rates being 76 and 76. At this time there was slight horizontal nystagmus of the fundus toward the subject's left. The retinal arteries were slightly constricted. Both arteries and veins seemed smaller than at the previous examination. At times it was difficult to see the fundus properly. The boat continued to roll somewhat.

At 9.25 no vessels were visible in the ear drums. The subject felt well in every way. At 9.42 the blood-pressures were 135, 130, 135, the pulse-rates being 80, 84 and 80. At 10.09 the retinal arteries were somewhat constricted, the veins appearing large, dark and full. Occasionally there were nystagmic jerks of the fundus toward the subject's left. At 10.12 the stomach contents were removed, and the stomach irrigated. The amount of chyme recovered was ℥iv. For the purpose of removing the stomach contents, the subject was hypnotized, as his pharynx could not tolerate the stomach-tube. On previous occasions this subject, even under hypnosis and with appropriate suggestions, could not tolerate the stomach-tube. On this occasion, however, it was suggested to him, both before and during hypnosis, that the operation would cause no discomfort and little difficulty was experienced in evacuating and irrigating the stomach. When the stomach irrigation had been completed, the subject was allowed to remain in hypnosis, and at 10.30 an Ewald meal was given with the suggestion that it was cake and champagne. During the meal, and at frequent intervals afterward, it was suggested that the motions of the boat would not cause seasickness or the usual disagreeable phenomena. At 10.51 the boat was rolling considerably, and there was marked nystagmus of the fundus to the left. To the naked eye at this time there was nystagmus to the left, when the subject turned his eyes to the left, and to the right when he turned his eyes to the right. In an observation made on a subsequent occasion on shore, it was found that when the subject turned his eyes to the left the latter jerked slightly with the watch, whilst with the eyes turned to the right the eyes jerked to the right and with the watch. At 11.03 the retinal arteries were moderately dilated, the veins being full and dark. There was slight nystagmus of the fundus to the subject's left, but at times the oscillations were simply back and forth movements of about equal rate and range. At this time the boat was rolling so much that subject "B" felt rather dizzy and heavy in the head. At 11.22 the retinal arteries were constricted, the veins being dark and full. There was horizontal nystagmus of the fundus to the left. The boat, lying at anchor, was rolling with sickening effect. At 11.35 the stomach was evacuated and irrigated. The amount of chyme recovered was ℥ivss. At 1.05 p.m. the retinal arteries were constricted. The retina looked pale, and there was nystagmus of the fundus to the subject's left. At this time the subject felt very dizzy. In fact he was dizzier and sicker under hypnosis than in the ordinary waking state. After irrigation of his stomach he slept considerably and could not be kept from lying down. At 1.09 the blood-pressures were 125, 125, the pulse-rates being 68 and 68. The boat still continued to rock, and the subject felt dizzy and heavy. At 1.25 there was experienced heaviness in the left side of the head, and the subject felt hungry, sleepy, and tired. No vessels were visible in the membrana tympani. At 2 the subject, on being taken out of hypnosis, complained of hunger, but on being taken to the dining-room, a surprisingly small amount of food soon satisfied his appetite.

During hypnosis, on attempting to walk, the subject almost fell down the companion-way. It seemed that his powers of equilibration had become considerably disordered.

Analysis of specimen taken from subject "F," Jan. 8th, on board the *Angler*. Ingested at 9.09 a.m. an Ewald breakfast. Removed at 10.12. Amount of chyme, ℥ivss. Macroscopically the chyme was normal in appearance. Reaction (litmus) acid. Resorcin test showed the presence of free HCl.

Tot. acidity, 94, or .343% by wt.
Free HCl, 80, or .292% by wt.
Comb'd HCl, 12, or .043% by wt.
Tot. HCl, 92, or .335% by wt.
Acid salts, 2, or .007% by wt.

Lactic acid, trace.
Starch, absent.

Amylodextrin, present.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, absent.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, moderate amount.
Blood, absent.

Absorption and motility tests not made.

Analysis of specimen taken from subject "F," Jan. 8th, on board the *Angler*. Ingested at 10.30 a.m. an Ewald meal. Removed at 11.35. Amount of chyme, ℥ivss. Macroscopically the chyme was normal in appearance. During the period that the meal was retained, the subject was in waking hypnosis and under the suggestion that the movements of the boat would not cause seasickness or the usual digestive disturbances. Reaction (litmus), acid. Resorcin test showed the presence of free HCl.

Tot. acidity, 66, or .240% by wt.
Free HCl, 48, or .175% by wt.
Comb'd HCl, 16, or .058% by wt.
Tot. HCl, 64, or .233% by wt.
Acid salts, 2, or .007% by wt.

Lactic acid, absent.
Starch, absent.

Amylodextrin, present.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, faint trace.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, moderate amount.
Blood, absent.

The K I absorption test reacted in 12 minutes. The salol motility test did not react within the hour.

The details of the tests made on subject "S" aboard the *Angler*, January 8th, were as follows: At 6 a.m. the subject had breakfast, consisting of bread and butter, with milk and coffee, "half and half," sweetened with sugar. At 8.08 a.m. the ear drums were normal, no blood-vessels being visible. The blood-pressures were 130 and 130, the pulse-rates being 76 and 76. At 8.20 the boat started. At 9 the stomach-tube was introduced, but nothing was recovered. At 9.38 an Ewald meal was given. At 10.38 the stomach contents were removed, and the stomach irrigated. The amount of chyme recovered was ℥iii. During the period that the meal was retained, the boat rolled moderately. At 10.48 the blood-pressures were 130, 130, the pulse-rates being 68 and 72. At this time the boat was rolling considerably, but the naked eye could discover no trace of nystagmus. At 10.55 the boat was still rolling badly, and when the subject turned his eyes to the right or left, a trace of horizontal nystagmus was visible. At 11.07 the blood-pressures were 125, 125, the pulse-rate being 68 and 72. At this time the boat was rolling and pitching considerably. At 11.08 the subject was given, *per os*, pyloric extract equivalent to the yield of 1¼ stomachs. This extract had been made on January 5th. At

11.52 the blood-pressures were 125, 125, 125, the pulse-rates being 64, 64 and 64. At this time the boat was lying at anchor, but rolling considerably. At 11.57 an Ewald meal was given. At 12.08 p.m. the boat, still at anchor, was rolling badly. The blood-pressures were 120, 115, 125, 180, the pulse-rates being 64, 64, 68 and 64. About this time the subject complained of cramps in the stomach, caused probably by the pyloric extract. At 12.57 the stomach contents were removed and the stomach irrigated. The amount of chyme recovered was ℥iiiss. At 1.28, the blood-pressures were 120, 115, the pulse-rates being 68 and 68. The patient was but little affected by the motions of the boat during the trip, and felt fairly well. At 1.25, on looking to the right or left, a trace of horizontal nystagmus was visible. There was no vertigo or sense of dizziness

Analysis of specimen taken from subject "S," Jan. 8th, on board the *Angler*. Ingested at 9.38 a.m. an Ewald meal. Removed at 10.38. Amount of chyme, ℥iii. Macroscopically the chyme was normal in appearance. Reaction (litmus), acid. Resorcin test showed the presence of free HCl.

Tot. acidity, 56, or .204% by wt.
Free HCl, 34, or .124% by wt.
Comb'd HCl, 16, or .058% by wt.
Tot. HCl., 50, or .182% by wt.
Acid salts, 6, or .021% by wt.

Lactic acid, absent.
Starch, absent.

Amylodextrin, absent.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, faint trace.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, moderate amount.
Blood, absent.

Absorption and motility tests not made.

Analysis of specimen taken from subject "S," Jan. 8th, on board the *Angler*. Ingested at 11.57 a.m. an Ewald meal. Removed at 12.57 p.m. Amount of chyme, ℥iiiss. Macroscopically the chyme was normal in appearance. At 11.08 the subject had received, *per os*, pyloric extract equivalent to the yield of 1¼ stomachs. Reaction (litmus), acid. Resorcin test showed the presence of free HCl.

Tot. acidity, 66, or .240% by wt.
Free HCl, 50, or .182% by wt.
Comb'd HCl, 14, or .051% by wt.

Tot. HCl, 64, or .232% by wt.
Acid salts, 2, or .007% by wt.

Lactic acid, absent.
Starch, absent.

Amylodextrin, present.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, faint trace.
Peptones, present.
Enzymes, normal.
Bile, present.
Mucus, moderate amount.
Blood, absent.

Absorption and motility test not made.

The details of the tests made on subject "B" aboard the *Angler*, January 8th, were as follows: At 4.45 a.m., the subject drank ℥viii of water. At 6.15 he had breakfast, consisting of bread ℥viii, butter ℥iv, and tea ℥xiv with milk and sugar. At 8.11 the blood-pressures were 135, 135, the pulse-rates being 100 and 104. At 8.20 the boat started. At this time the boat was rolling considerably, so that the subject felt a little dizziness and heaviness in the head. At 9.22 the subject complained of fulness and lightness in the head and of a band sensation about the lower portion of the thorax (hypochondriac region). At 9.30 the gastric contents were removed, and the stomach irrigated. The amount of chyme recovered was ℥viii. It contained much thick mucus, a few old blood-stained scales, and much fat. There was a distinct odour of lactic acid. At 9.58 an Ewald meal was given. At 10.38 the blood-pressures were 125, 125, the pulse-rates being 84 and 84. At 10.40 pyloric extract, equivalent to the yield of 1¼ stomachs, was given *per os*. At 10.42 the boat was rolling and pitching considerably. The subject felt heavy in the head, and his face was flushed and hot, though the rest of his body was cool. At 10.49 the boat was still rolling, and pitching badly. The subject, who was sitting upright with his coronal plane parallel to the long axis of the boat, exhibited no signs of nystagmus. At 10.58 the blood-pressures were 115, 115, the pulse-rates being 84 and 84. At 11.05 the boat was still rolling and pitching. The subject complained of heaviness in the head. The face was flushed and hot, whilst the stomach felt well. At 11.12 the blood-pressures were 125, 125, the pulse-rates being 88 and 84. The head still felt a little heavy, but the stomach felt well. At 11.15 an Ewald meal was given. At this time the boat, lying at anchor, rolled considerably and with sickening effect. At 11.30 the blood-pressures were 140, 145, 145, the pulse-rates being 84, 84 and 84. At this time the boat lay very still at anchor, and the subject felt very well. At 11.47 the boat, still at anchor, rolled considerably. The subject felt a sensation of warmth in the stomach. There were some eructations. At 11.57 the blood-pressures were 130, 130, the pulse-rates being 80 and 80. At 12.11 p.m. the boat was rocking considerably, and the subject's head felt heavy. At 12.14 the boat was steady. At 12.15 the gastric contents were removed, and the stomach irrigated. The amount of chyme recovered was ℥iii. It contained much mucus. At 12.41 the subject complained of pains in the lower abdomen. At 12.50 the blood-pressures were 115, 115, the pulse-rates being 80 and 80. At this time the boat was still at anchor and rolling considerably. At 1.18 the blood-pressures were 120, 115, the pulse-rates being 80 and 80. The boat was rolling slightly at this time, and the subject had a burning sensation referred to the epigastrium, and a slight ache in the top of the head, with a sense of fulness in the latter. At 1.25 on turning the eyes to the left, there was slight horizontal nystagmus to the left. On looking to the right there was no visible nystagmus.

Analysis of specimen taken from subject "B," Jan. 8th, on board the *Angler*. Ingested at 6.15 a.m. breakfast consisting of bread ℥viii, butter ℥iv, tea ℥xiv, with milk and sugar. Removed at 9.30. Amount of chyme, ℥viii. The chyme contained much thick mucus, a few old blood-stained scales, and a quantity of fat. There was a distinct odour of lactic acid. Reaction (litmus), acid. Resorcin test showed a feeble reaction for free HCl.

Tot. acidity, 38, or .138% by wt.
Free HCl, 18, or .065% by wt.
Comb'd HCl, 18, or .065% by wt.
Tot. HCl, 36, or .130% by wt.
Acid salts, 2, or .007% by wt.
Lactic acid, present.
Starch, absent.
Amylodextrin, absent.
Erythrodextrin, absent.
Maltose, trace.
Albumin, trace.
Peptones, present.
Peptonizing enzymes, diminished.
Peptonizing zymogens, normal.
Coagulating enzymes, normal.
Bile, absent.
Mucus, in excess.
Blood, trace.

Absorption and motility tests not made.

Analysis of specimen taken from subject "B," Jan. 8th, aboard the *Angler*. Ingested at 11.15 a.m. an Ewald meal. Removed at 12.15. Amount of chyme, ℥iii. The chyme contained a considerable amount of thick mucus and a few blood-stained flakes. At 9.58 an Ewald meal had been taken and retained permanently. At 10.40 pyloric extract, equivalent to the yield of 1¼ stomachs, had been taken *per os*. Reaction (litmus), acid. Resorcin test showed the presence of free HCl.

Tot. acidity, 44, or .160% by wt.
Free HCl, 22, or .080% by wt.
Comb'd HCl, 20, or .073% by wt.
Tot. HCl, 42, or .153% by wt.
Acid salts, 2, or .007% by wt.

Lactic acid, absent.
Starch, absent.

Amylodextrin, present.
Erythrodextrin, present.
Achroödextrin, present.
Maltose, present.
Albumin, trace.
Peptones, present.
Enzymes, normal.
Bile, absent.
Mucus, in excess.
Blood, trace.

Absorption and motility tests not made.

The urine after the tests had a specific gravity of 1018 and did not react for albumin, sugar, glycuronic acid or phosphates.

CHAPTER XXIX

GENERAL CONCLUSIONS FROM STUDIES IN SEA-SICKNESS. PROTOCOLS

The general conclusions from the foregoing observations are as follows:

1. The effects of seasickness upon the organism as a whole, and upon the mechanisms of the circulation and digestion are quite analogous to those of the sickness caused by rotation, aural irrigations, and by galvanism applied over the mastoid areas.

2. Disturbances of the circulation are not the primary cause of seasickness since the chief subjective phenomena of the malady are found present, simultaneously with both efficient and impaired states of the circulatory mechanisms. Disturbed circulation, however, especially vaso-motor exhaustion, is frequently associated with seasickness and is an important feature of the malady.

3. Digestive disturbances, similarly, are not the primary cause of seasickness, though they are constant accompaniments and frequently the most salient and distressing features of the condition. The firm and persistent closure of the pylorus and the lowered acidity, as seen in the analyses of the gastric contents, are identical with the phenomena observed in rotations and aural irrigations. In extreme conditions, such as those encountered on the *Lady Wolseley* and on the *Quatre Frères*, there was complete absence of acids and digestive enzymes. Prolonged exposure to conditions that cause seasickness are undoubtedly injurious to the digestive organs, and more especially to the stomach which may become seriously impaired in its functions and even the seat of organic disease. Thus subject "B" undoubtedly developed during the various journeyings a mild grade of chronic gastritis. The irregu-

lar mode of life and the alcohol taken by him during the period extending from July 10th to July 27th, were probably factors in the causation of his gastritis, but the continually repeated perturbations of the nervous mechanisms controlling the general circulation as well as gastric secretion and motility were the chief cause.

4. Disordered states of the circulatory and digestive mechanisms when once initiated become powerful secondary sources of irritation. Thus injudicious muscular exertion, the retention of stagnant gastric contents or the introduction of improper food into the rebellious and highly irritable stomach tend greatly to over-irritation and exhaustion of the gastric vagal, vaso-constrictor and other centres.

5. The ventilation and temperature of the cabin are also important secondary factors in seasickness, the cool, well-ventilated room tending greatly to restore the individual, whilst a hot stuffy cabin may cause disturbances of the circulatory and digestive mechanisms as distressing almost as those which occur in seasickness though caused primarily in another way.

6. Cold bathing and the employment of methods or drugs calculated to enhance directly vaso-motor efficiency by stimulation of the nervous centres are contraindicated where the circulatory depression is due to exhaustion from over-irritation. Muscular exertion in these conditions is therefore injurious. At a later stage, when there is evidence of restoration of function on the part of the nervous centres, and when there is reason to believe the stage of exhaustion is past, mild exercise, massage and hydrotherapy are of undoubted benefit. The last of these measures, however, has to be resorted to with the greatest caution and only after special study of the peculiarities of the individual's circulatory mechanisms. For even more imperative reasons drugs and procedures that tend to depress or over-stimulate the vaso-motor mechanisms, or indeed any of the medullary centres, are contraindicated. Drugs that depress the psychic and higher centres, such as bromides and alcohol, are on the whole of questionable benefit and alcohol in excessive quantities is distinctly harmful to the circulation. The effects of morphin, cocain, hyoscin, and nitroglycerin in rotation

sickness were such as to offer little hope of usefulness in seasickness. They were, therefore, excluded from the list of possible safe and beneficial measures.

7. Psychic depression, and disagreeable sights and odours are also important secondary causes of distress. Hence sunshine and clear weather with pleasant surroundings and agreeable companionship are of benefit.

8. Since exhaustion of the nervous centres is characteristic of seasickness at its height, mental effort, especially when coupled with the use of the eyes as in reading, may be particularly harmful. Quite frequently during the progress of recovery from an acute spell of seasickness subject "B" has retarded his recovery by thinking too intently over his future plans or by emotional reading or by indulging in a train of emotional thought.

9. Recovery from seasickness means the adaptation of the individual's organism to the rhythm and extent of the movements of that particular boat upon which he has been travelling. Hence a traveller may have recovered from seasickness upon an ocean liner and later fall a victim to the motions of a Channel steamer. Even marked aggravation of the movements of the boat upon which an individual has recovered may cause a return of all the phenomena of seasickness. This frequently occurs under varying conditions of weather.

Frequently individuals who were for the greater part of their lives victims of seasickness lose their susceptibility. On the other hand persons who have been immune may, at some time or other, develop a susceptibility for the malady. This is one of the mysteries which sailors and others hurl at the medical man who presumes to know anything about seasickness. However, bilateral degeneration of the eighth nerve which so frequently occurs in advancing years and earlier in certain families, accounts for the first class of cases. If the degeneration progresses irregularly so that the nerve on one side alone is affected or is affected to a greater or less extent than the nerve on the other side, we have a condition of relative heightened irritability of the vestibular nerve endings in one labyrinth which, as we have seen under aural irrigations, is the great cause of disturbance of the vestibulo-cerebellar mechanisms involved in equilibration. Actual cases of this kind have been studied, but the details will be related in another chapter.

10. Atropin, more especially in combination with strychnin, is effective in combating the subjective symptoms of seasickness, especially the nausea and the gastric and cerebral discomfort, but it has no direct effect in promoting gastric secretion and digestion, and does not prevent the incoordination or disturbances of equilibrium incidental to seasickness although it tends to eliminate the associated sense of vertigo.

11. Although the tests made on January 8th afford, in themselves, no satisfactory basis for conclusions as to the effect of hypnotic suggestion in seasickness, the results obtained, especially when taken into consideration with the results obtained from hypnotic suggestion in aural irrigations and rotations, indicate that hypnotic suggestion is not a very efficient means of offsetting or preventing the effects of seasickness even during actual hypnosis, not to mention the rapidly waning influence of post-hypnotic suggestion. It is to be regretted that further opportunity has not so far presented itself for the study of this important phase of the subject.

12. The fact that extracts made from the various portions of the gastric mucous membrane were about equally efficient (in some instances the "intermediate extract" appearing to be the most efficient of the three) in stimulating the flow of normal gastric juice seems to indicate that the effects of the extracts were due to the contained substances with secretogogue action rather than to gastric secretin developed by decoction. This fact was more apparent in subsequent tests made on shore and reported elsewhere. In these tests it was found that ordinary broth was as efficient, and at times more so than decoctions of pyloric or fundic mucous membrane. Following the administration of these extracts no appreciable alterations in blood-pressure or pulse-rate were observed which could be safely attributed to the extracts. It seems reasonable, therefore, to conclude that gastric secretin is inert when administered *per os* (possibly, because of changes induced as the decoction reaches the stomach) and that the effect of decoctions of the gastric mucous membrane given *per os* in promoting the flow of normal gastric juice is due to

other substances (secretogogues) contained in the decoctions. In the matter of enhancing the flow of gastric juice, therefore, decoctions of gastric mucous membrane have, when administered *per os* no advantage over ordinary meat-extracts, such as beef tea, soup, etc. Decoctions of gastric mucous membrane seem however to aid absorption and to promote, to some extent, gastric and intestinal motility.

STUDIES IN SEASICKNESS

Protocol 1.—Observations made on subject "B" aboard the steamship "Taurus" Dec. 27, 1908. Temperature of air 60° F.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
8:25	96	120	On boat before starting.
	96	120	" " "
8:37	92	115	After start. Slight roll. Weather calm.
	92	120	
8:45	80	110	Slight headache.
9:30	76	105	"
10:00	68	105	Pulse slightly irregular.
10:30	68	95	Headache. Face flushed.
11:05	68	105	Conjunctivæ slightly congested.
	72	100	
12:30	68	110	At anchor.
	64	110	"
	64	110	"

Protocol 2.—On "B" aboard steamer "Angler," April 4, 1909.

a.m.			
8:05	88	105	On boat after slight injury of hand.
8:10	84	110	Feels well in fresh, cool air.
8:11	80	115	Boat started.
	88	125	
	80	125	
8:20	80	120	Conjunctivæ slightly congested.
8:22	80	120	Feels well. Draught on right shoulder.
8:26	84	125	Dizzy; slight lump-sensation; fulness in head.
8:28	80	125	Well again, but some fulness in head still.
8:29	80	120	" " " " "
8:32	88	125	Feels well, but has fulness in head and is slightly dizzy.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
8:33	76	125	Taking deep breaths. Conscious of heart action after slight exertion, viz.: standing on seat.
	76	120	
8:35	72	120	Frontal headache. Fulness in head. Conjunctivæ congested.
8:37	80	120	Face pale. Slight nausea; stomach distress.
8:38	76	125	Draught on shoulder. Feels as if about to sneeze.
8:39	76	120	Dizzy and mentally depressed.
8:40	72	120	" " "
8:41	80	125	Feels well. Sight of water rushing by distresses.
8:43	72	120	Cold on one side because of draughts.
8:44	76	120	Face pale. Conjunctivæ congested. Pupils normal.
8:49	72	120	Face pale. Cheeks warm. Feels well. Eructations.
8:51	76	125	Artery contracted. Feels depressed.
8:56	72	125	Lump-sensation. Fulness in head. Eructations.
8:58	76	120	Nauseated. Sense of fulness in ears. Boat steady.
9:00	76	125	Conscious of respiratory movements.
9:01	72	125	Sense of tension in scalp over occiput. "Lump-sensation" in stomach.
9:03	76	125	Abnormal sensation (fulness) in left ear.
9:04	76	125	Head heavy and full. "Lump." Dizzy. Uninterested.
9:06	76	125	Taking deep breaths. Nauseated. Not so depressed now.
9:12	76	125	"Lump." Sense of weight in frontal region. Conscious of stomach contractions.
9:13	72	25	Eructations. "Lump." Nausea.
9:15	76	125	Moving head from side to side aggravates nausea.
9:17	72	125	Dizzy as boat lurched.
9:25	80	120	"Lump." Dizzy. Arteries contracted.
9:41	72	125	Sick feeling in head. Deep breaths. Noise of water distresses.
9:46	68	125	Fulness about ears as if head was in the grip of something.
9:52	68	125	"Lump." Eructations. Boat rolling. Saliva increased.
9:55	68	120	Conjunctivæ congested. Face not so pale now. Yawning.
9:57	68	120	Boat rolling and pitching. Dizzy. Arteries contracted. Deep breaths. Yawning. Eructations.
10:00	68	120	Tobacco-smoke inoffensive to smell. Subject has been out on bow.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
10:02	68	120	Photophobia. Sneezed. Muscular tremors. Dizzy. Difficulty in balancing. Mental effort causes distress in head.
10:03	68	120	Arteries contracted moderately.
10:11	68	130	Feels well. Slight "lump." Arteries contracted.
10:13	68	135	Arteries much contracted.
10:15	68	125	Feels well.
10:17	72	125	Arteries contracted. Conjunctivæ congested. Pupils normal.
10:20	64	125	Sense of weight in head. Boat rolling and pitching.
10:22	64	130	Arteries contracted. Deep breaths. Frontal headache.
10:24	72	125	Lightness in head. Feels well.
10:37	64	125	Boat stopped. Feels well. Air cold.
10:38	68	135	Tobacco-smoke not disagreeable but not pleasant.
10:42	64	125	At anchor. Fulness in head.
10:10	64	125	Slight eructations. Deep breaths.
11:15	64	125	
12:00	68	115	Feels well. Hands cold. Face flushed and warm.
12:04	68	125	Feels well.
1:40	72	120	Ate an orange. Feels refreshed. Boat rolling.
1:43	68	125	Fulness in head. Arteries moderately contracted.
1:46	68	125	Strolled on deck.
2:22	68	120	Feels well. Pupils and conjunctivæ normal.
2:25	68	125	Artery moderately large. Stomach and head feel well.
2:27	68	125	Feels well in every way.
2:28	64	125	Weighed anchor. Boat rolling some.
2:30	68	125	Fulness and lightness in head. Feels fairly well.
2:47	68	125	Before starting homeward feels well.
2:49	68	125	Not yet started. Artery normal.
2:52	72	125	Boat rolling. Fulness in head.
2:58	68	125	Artery contracted somewhat. Feels well.
3:01	64	125	
3:03	68	125	
3:04	64	120	Boat started homeward. Eructation.
3:05	64	120	Headache. Face flushed. Feels well.
3:06	64	120	Fulness in head. Face hot.
3:09	64	120	Sense of weight in head. Feels well.
3:12	68	120	Sight of passing water distresses.
3:30	68	125	Odour of tobacco-smoke slightly disagreeable.

Protocol 3.—On "B" aboard steamer "Angler," May 30, 1909.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
7:46	80	120	On boat resting.
	76	120	"
	76	120	"
	76	120	"
	72	115	"
	76	120	"
	76	120	"
	76	115	"
	76	120	"
	76	120	"
	76	120	"
	76	125	"
	76	120	"
	76	125	"
8:14	..	...	Boat starts. Weather fine. Boat steady.
8:15	72	120	
	72	130	
	80	125	
	76	115	
	72	120	
	72	120	
8:21	..	...	Ewald breakfast.
8:22	76	120	
	76	125	
	80	120	
	76	120	
8:45	76	120	"Lump-sensation." Saliva increased.
	72	120	Fulness in head. Eructations.
	76	120	
	72	120	
	72	120	
	72	115	
	72	115	
9:08	76	110	Dizzy. Fulness in head. Saliva increased.
	72	110	
	72	110	
	72	105	
9:14	72	110	Frontal headache. "Lump-sensation" in stomach.
	72	110	
	72	110	
	72	105	
9:19	76	110	General headache. Fulness in top of head.
	72	105	
	68	115	
	76	115	
	76	110	Ache over eyes. Pain in eyeballs.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	76	115	"Lump-sensation."
	72	115	
9:32	76	115	Boat rolling some. Headache. Eructations.
	72	115	
9:35	76	115	Eructations. Disinclined for work.
	72	110	
	68	110	
	68	110	
9:53	68	115	Frontal headache.
9:55	72	110	Dizzy. Lump-sensation.
	76	115	
	72	115	
	68	115	
9:59	72	110	Feels as if about to sneeze. General headache.
	72	115	Disinclined for work. Would like to lie down.
10:14	..	...	Boat at anchor.
10:24	64	115	Stomach contents withdrawn.
	76	120	
	76	120	
	80	120	
10:32	76	120	Headache.
	76	120	
	76	120	
10:36	76	120	Boat rolling at anchor.
	76	120	
	76	120	
	72	115	
	72	110	
	72	110	
	72	110	
	68	110	
10:47	72	110	Diffuse headache. Looks worried.
	68	105	Boat still at anchor and rolling.
	76	105	
	72	110	
	68	110	
	76	115	
10:54	72	110	Lunch of beef sandwiches and oranges.
	72	110	
11:21	80	110	Boat starts. Much rolling.
	72	110	Dizzy. Fulness in head.
	72	110	Headache.
	76	110	
	76	110	
	72	115	
	72	110	
	72	105	
	76	115	
	72	115	

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	68	115	
	72	115	
11:35	76	115	Boat at anchor. Walked deck from 11:35 to 12:00.
12:00	72	115	Boat rolling at anchor.
	72	110	Headache. Arteries contracted.
	72	110	
	72	105	
	80	115	Annoyed by intermeddler.
	76	115	
	76	115	
	76	115	
	80	110	
	76	110	
	76	115	
	76	115	
	76	110	
p.m.	76	115	
12:22	76	115	Boat starts. Headache. Boat rolling much.
	76	120	Sight of moving water distresses the eyes.
	76	115	
	72	110	Dizzy. Headache.
	76	115	
	76	120	Pain in back of the neck on right side.
	80	120	
	76	120	
	76	125	Arteries contracted.
12:31	76	120	Pain in left of epigastrium.
	84	125	Headache.
	80	125	Pain in eyeballs.
	80	120	Boat rolling and pitching badly.
	80	120	
	80	120	
12:38	80	120	Boat at anchor again.
12:39	76	120	Rolling at anchor.
	80	120	Pain in epigastrium.
	76	115	Sense of constriction about lower part of chest.
	80	115	
	80	115	
	76	110	
	76	110	
12:46	76	115	Dizzy. Headache.
12:59	80	110	After walking about.
	76	110	"Lump-sensation."
	76	110	Headache occipital and frontal.
	80	110	
	80	110	
	80	105	
	80	115	

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	76	110	
	80	95	
1:10	80	100	Arteries dilated.
	80	100	
	76	105	
	76	105	
	80	105	Fulness in head. Nausea.
	80	105	
	80	95	Arteries dilated.
	80	90	
	80	90	
	80	90	Pain in epigastrium.
	80	90	Sense of band about lower part of chest.
	80	100	
1:22	80	90	
	80	90	
	80	95	
	80	105	Biparietal headache. Feels very tired.
	80	105	
	80	95	
	80	95	
	76	85	
1:31	80	95	
	80	95	
	76	95	
	76	95	Headache. Dizzy.
	76	105	
	76	100	Rolling much.
	76	110	Arteries contracted.
	76	110	
	76	110	
	72	110	Headache. Dizzy.
1:44	76	115	Annoyed by intermeddler.
	80	110	Angry and irritable. Muscles trembling all over.
	76	115	
	80	100	
	80	95	
	80	95	Severe headache.
	76	100	Eructations. Stomach feels well.
	80	100	
1:53	80	95	Headache.
	80	95	
	80	100	
	80	100	
	80	90	Taking deep breaths.
	80	100	Frontal headache.
2:00	84	90	Headache. Eructation.
	76	90	Stomach feels well.
	80	95	

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Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
2:05	80	95	Boat starts.
	80	100	
2:07	80	100	Boat stops.
	84	100	Feels weak. Perspiring freely.
	84	95	Eructation. Arteries dilated.
	84	95	Tired and weak.
	84	95	Would like to rest head on something.
2:27	76	100	Walked about from 2:14 to 2:27.
	76	100	Felt weak and tremulous in muscles.
	72	110	General wretchedness.
	72	110	Boat steady and still at anchor.
	76	110	
	80	110	
2:35	84	115	Boat started for home.
	76	105	
	80	95	
	80	100	
	80	100	
	76	90	
	80	100	Headache.
	80	90	Taking deep breaths.
	76	90	
	80	95	
	80	100	
	80	100	
	80	100	
	80	100	
2:55	76	100	Headache.
	76	100	Odour of tobacco-smoke offensive.
	80	100	
3:00	80	100	
	76	100	Boat moving smoothly.
	80	105	Fresh cool breeze. Headache.
	72	100	
	76	100	
	72	100	
	76	100	
	76	105	
	76	105	
3:15	80	100	Cool breeze on occiput.
	76	100	
	80	95	Headache persists.
	80	95	
	80	95	
	80	90	
	76	110	
	80	110	Arteries contracted.
	80	110	Feels better and brighter.
3:23	76	110	Boat going smoothly.
	76	110	Headache almost gone.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	84	105	
	80	100	Headache bad again.
	80	100	
	80	100	Pain through eyeballs.
	76	100	
3:30	80	110	
	72	110	Headache.
	72	110	Not so tired now.
	84	110	
	76	105	Occipital headache.
3:36	80	100	
3:44	68	110	After walking in the open air.
	76	100	
	80	110	Throbbing frontal headache.
	76	100	
	72	110	
	76	110	
	72	100	
3:50	76	95	Coronal headache.
	76	110	
	76	110	
3:55	76	105	Pain in back of neck, right side.
	76	105	
	76	100	Arteries contracted. Face pale.
	80	100	Deep breaths.
	76	105	Pain in occiput and right side of neck.
4:00	76	105	Feeling wretched.
4:01	80	110	Right hemicrania, especially over right, superior curved line of occipital bone.
	76	105	
	76	105	
4:05	80	105	Taking deep breaths.
	76	105	Dull ache in eyeballs.
	76	100	Looking at things causes occipital ache.
	76	100	
	76	95	
4:10	80	100	Pain all over scalp, but worse on right side.
	72	105	
	80	100	Sweating. Eructations.
	76	100	Would like to lie down.
	80	100	Would like to let eyelids droop.
4:15	80	100	Closing eyes causes distress.
	76	100	
	76	100	
	76	100	
	80	100	Lump-sensation in stomach.
	72	105	Eructations.
	76	100	Headache in right parietal region.
4:21	76	100	
4:26	76	110	After walking about.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	72	110	
4:28	72	110	Headache across top of head.
	76	110	
4:30	76	100	Lump-sensation.
	72	105	Deep breaths.
4:32	76	105	Occipital headache.
	76	105	Sweating. Feels weak.
	76	105	Lump-sensation marked.
	76	110	
4:35	76	105	Coronal headache.
	76	115	
	76	105	
4:38	76	110	Headache not so bad.
	76	100	Subject brighter and more cheerful.
	76	100	
	76	100	Lump-sensation persists.
	80	100	Taking deep breaths. Sinking feeling in stomach.
4:42	72	115	Arteries contracted.
	80	110	
4:44	80	100	Arteries dilated.
	80	100	Lump-sensation. Feels sick.
	80	110	Occipital headache in region of hat band.
4:46	76	110	
4:47	..	...	Just before landing. Some congestion around the periphery of drum membranes and along the handle of the malleus.
6:36	..	...	Occipital headache persists, i.e., nearly two hours after landing.

Protocol 4.—On "B" aboard the motor launch "Maggie," May 31, 1909. Trip lasting 1 hour.

a.m.			
5:25	96	105	Standing after hard day's work.
	96	105	Feet heavy and tired.
	96	100	
	72	130	Lying supine. Face flushed. Pupils moderately dilated. Room dark.
	80	125	Lying supine. Face flushed. Pupils moderately dilated. Room dark.
	84	120	Lying supine. Face flushed. Pupils moderately dilated. Room dark.
	76	120	Lying supine. Face flushed. Pupils moderately dilated. Room dark.
	80	120	Lying supine. Face flushed. Pupils moderately dilated. Room dark.
	84	120	Lying supine. Face flushed. Pupils moderately dilated. Room dark.

Pulse-rate.	Blood-press.	Remarks.
84	120	Lying supine. Face flushed. Pupils moderately dilated. Room dark.
108	105	Standing. Pupils normal but illumn'n better.
88	105	" " "
92	105	" " "
96	105	Boarded launch and started.
88	105	Fresh cool breeze. Vibration in feet. Feels well.
76	100	
84	105	
84	110	Arteries small. Feels well. Water smooth.
88	110	" " "
84	110	Feels fresh and cool in breeze.
88	95	Taking deep breaths.
92	95	Slight feeling of distress in head.
92	95	Perspiring along spine.
88	95	Lump-sensation. Feels a little sick.
88	95	" "
88	95	" "
88	90	" "
88	115	After turning north. Boat pitching much.
88	100	Lump-sensation. Taking deep breaths.
88	95	Head feels well.
88	105	Arteries contracted. Head feels cool.
88	105	Slight lump-sensation. Feels well.
88	110	Arteries contracted.
84	100	Lump-sensation.
84	95	Slight distress in head.
80	100	Lump-sensation. Eructations.
84	95	Arteries small.
84	95	
80	95	
80	95	Cool breeze. Feels well but has lump-sensation.
88	95	Vibration does not annoy now.
80	90	Arteries much contracted.
76	95	Taking deep breaths.
80	95	Feet tired, heavy and aching.
80	95	Lump-sensation.
80	100	" Taking deep breaths.
80	105	Just before landing.
80	105	Landed.
76	115	1 min. after landing.
92	115	2 " Feels well.
80	115	Drank ginger ale, ℥viii.
84	120	1 min. later.
88	115	2 "

Protocol 5.—On "B" aboard steamship "Caledonia." Transatlantic trip commenced June 26, 1909.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
2:07	84	130	Lying, after exertion, on board *Caledonia.*
	84	125	"
	76	125	"
	72	125	"
2:22	72	125	Boat starts.
	84	120	"
	88	115	Standing.
2:23	84	115	"
	88	115	"
2:55	68	115	Lump-sensation in stomach.
3:09	72	115	Lying. Odour of tobacco-smoke offensive.
	72	110	" Saliva increased.
	72	110	" Occipital headache. Arteries small.
	72	110	" Ache behind right mastoid.
3:20	76	95	Standing. Feels well. Fresh breeze from port-hole.
	76	95	Standing.
	84	100	"
	80	100	" Head feels well.
	88	95	" Slightly hungry.
	80	95	" Eructations.
3:30	80	90	Standing. Masklike feeling in scalp.
4:40	84	100	" Tobacco-smoke not offensive.
	84	100	" Slightly dizzy.
4:44	80	100	"
4:45	68	110	Lying. Arteries dilated.
	72	110	" "
	68	110	" "
	68	110	"
	68	105	"
	68	105	"
	68	105	"
	68	105	"
4:55	80	90	Standing. Pulse-rhythm irregular. Arteries small.
	76	95	Standing.
	84	95	"
5:00	84	100	"
5:19	88	90	" Slightly dizzy and faint.
5:21	80	95	Standing.
	80	95	"
7:00	..	...	Dinner. K I aborption test reacted in 2 hours 17 min. Salol motility test reacted in 2 hours 22 min.
7:23	76	115	Lying.
	72	115	Feels well. Lying.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	72	115	Lying. Conscious of stomach movements.
	72	115	" Slight lump-sensation.
	72	125	" Head feels well.
	72	125	"
	76	125	"
	72	120	"
	72	115	"
	72	115	" Arteries larger.
	76	115	"
	80	115	" Slight lump-sensation.
	76	120	"
	76	115	" Saliva free all through K I tests.
	76	115	"
	72	115	"
	72	115	" Biparietal headache.
	72	115	" Artery large.
	76	115	Lying. Marked "lump-sensation" in stomach.
	68	115	Lying.
	76	115	"
	72	115	"
	72	110	"
	76	115	" Lump-sensation marked.
	72	115	
8:14	84	115	"
8:15	84	95	Standing. Eructation. Flatus.
	88	100	" Momentary lightness in head.
	88	90	" Lump-sensation not so manifest.
	88	100	"
8:21	84	100	"
8:32	68	120	Lying. Lump-sensation in throat and stomach.
	68	115	Lying. Port-hole has just been closed.
	76	115	"
	68	115	"
8:39	72	115	"
8:40	88	95	Standing. Sweating. Cabin warm.
	92	85	"
	84	85	"
	84	95	"
8:56	76	95	
10:41	..	...	Went to bed. Slept well but dreamed much.

Protocol 6.—On "B." Second day at sea on "Caledonia." June 27, 1909.

a.m.			
6:44	72	105	Lying in bed before arising.
	72	105	"
6:46	68	110	"

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
6:47	88	90	Standing. Feels well.
	84	100	“ Cool breeze from port-hole.
6:49	84	105	“
7:05	88	95	“ 3 min. after cold plunge.
	84	95	“
7:07	88	95	“
7:40	68	115	Lying. Feels well. Face flushed.
	72	115	“ Pupils moderately dilated.
	72	120	“ Conjunctivæ normal.
	68	130	“
	68	125	“
	72	125	“
7:47	68	125	“
7:55	84	105	Standing.
	84	100	“
	80	105	“
	76	95	“
7:59	72	100	“
9:56	92	105	“ Feels well, but is a little tired.
	88	100	“
	92	95	“
10:04	92	95	“
10:05	76	120	Lying. Congestive headache.
	80	120	“ Pupils slightly dilated.
	80	120	“ Slight lump-sensation.
	76	120	“ Right frontal headache.
	76	125	“
	80	120	“ Slight stomach distress.
10:11	80	120	“
10:18	76	115	“ Slight headache. Arteries dilated.
10:20	80	90	Standing. Eructation. Lump-sensation less.
10:22	92	95	“
p.m.	92	95	“ Arteries moderately dilated.
12:21	76	110	“ After walking in cool breeze.
	76	105	“ Lump-sensation in stomach and throat.
	76	105	Standing.
	76	110	“
12:25	72	105	
12:57	72	95	“ After making analysis in cabin.
	72	95	“ Arteries small.
1:00	72	95	“ Lump-sensation.
1:01	68	110	Lying. Fulness in head. Artery moderately large.
	68	105	Lying.
	64	105	“
	68	115	“
1:07	68	110	“
1:08	72	85	Standing. Blood-pressure rose to 120 from effort, but instantly fell to 85.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	72	90	
1:10	76	95	Standing.
2:02	..	...	Dinner. K I absorption test reacted in 21 minutes.
4:15	68	105	Standing after walking in cool breeze.
	68	100	" Arteries small.
	72	100	"
4:21	68	100	"
4:36	68	100	" Arteries small. Feels well.
	68	100	"
	64	100	"
	64	95	"
4:41	68	100	"
4:42	68	110	Lying. Feels well.
	64	110	"
	60	115	"
4:45	68	120	"
4:46	60	115	" Fulness in head.
	60	115	" Slight headache.
	64	120	" Lump-sensation in stomach.
	60	115	" Right occipital pain.
4:56	64	115	" Feels sleepy.
4:57	64	130	On standing up.
	68	105	Standing. Feels a little better standing.
	68	100	"
5:00	68	100	"
8:50	68	90	" After walking in the air.
	72	95	" Feels well, but is tired.
8:52	68	95	"
8:53	64	105	Lying. Face flushed and hot.
	60	105	" Conscious of respiratory movements.
	60	115	" Lump-sensation in stomach.
	60	110	" Pupils slightly dilated.
	60	110	
	60	110	" Conjunctivæ normal.
9:03	60	115	" Feels sleepy.
9:04	72	125	On standing up. Blood-pressure instantly fell to 90.
	72	90	Standing. Sleepy.
	68	95	" Arteries moderately large.
	72	95	" Lump-sensation occasionally.
9:08	72	90	
9:35	..	...	Went to bed. Was awakened by indigestion, which soon disappeared.

Protocol 7.—On “B.” Third day on “Caledonia.” June 23, 1909.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
7:05	80	115	Lying in bed.
	64	115	“ “
7:07	64	115	“ “
7:08	80	105	Standing.
	80	110	“
7:11	80	105	“
7:12	80	95	“ Before cold tub.
7:13	72	140	Lying after getting into cold tub.
7:13½	72	135	“ Still in cold tub.
8:00	72	105	Standing after dressing.
8:01	76	100	“ Feels well. Arteries small.
8:02	68	120	Lying.
	64	125	“
	64	120	“
8:05	64	120	“
	80	110	Standing. Arteries small.
	76	105	“
	76	110	“
p.m.			
1:07	72	100	“ after sitting in the air.
	72	100	“
1:10	72	100	“
1:11	64	105	Lying.
1:14	64	110	“
1:15	64	110	Standing. Feels well.
	76	110	“
	72	100	“
1:17	72	115	“
1:56	76	110	“ after dinner.
	76	115	“
	80	105	“
1:59	76	105	“
3:40	68	105	“ after being on deck.
	76	110	“
	76	100	“
	76	100	“
	80	120	“ Sneezed.
	80	120	“
	80	120	“
	80	110	“
	76	105	“
3:55	76	105	“
3:56	72	115	Lying. Face flushed and hot.
	72	115	“ Feels sleepy.
	68	115	“ Conjunctivæ slightly congested.
	68	115	“ Pupils slightly dilated.
4:00	64	105	“ After falling asleep.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
4:01	64	100	Lying. Partially roused.
	64	105	" Asleep; face flushed.
4:10	64	105	" "
	60	110	" "
	60	105	" "
	60	105	" "
	64	105	" "
	64	110	" " Snoring. Face deeply flushed.
	64	110	" " " " "
	64	110	" " " " "
	60	105	" " Not snoring.
	60	110	" " Snoring. Face deeply flushed.
	64	105	" " " " "
	64	105	" Snoring. Face flushed.
	64	110	" " "
	64	110	" " "
	64	105	" " "
	64	105	" " "
4:26	64	105	" asleep.
4:27	60	110	" "
	60	110	" "
	64	110	" "
	60	105	" "
	60	105	" "
	60	105	" "
	60	105	" " Face flushed.
	60	105	" "
	60	105	" "
	60	105	" "
	60	105	" "
	60	105	" "
	60	105	" "
	60	105	" "
4:49	64	110	" " Snoring. Deep breaths.
4:50	60	100	" " " "
	60	105	" "
	60	105	" "
4:54	60	110	" "
	60	110	" "
4:58	72	110	" Awake.
	68	105	" Skin warm on awaking.
	68	105	"
5:08	68	110	" Half awake.
	68	110	" "
	68	110	" "
	68	110	" "
	68	110	" "
5:12	76	100	Standing. Cool breeze on body.
	72	100	" Arteries small.
	72	105	"

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
5:15	72	100	
10:23	76	105	Standing.
	76	100	"
10:25	72	100	"
10:26	68	115	Lying. Face flushed and hot.
	64	115	" Pupils moderately dilated.
	64	115	" Conjunctivæ normal.
10:28	64	115	"

Protocol 8.—On "B," aboard the "Caledonia." Fourth day. June 29, 1909.

a.m.			
7:00	76	120	Lying in bed. Cold breeze on arms.
	72	120	" " Weather foggy and cold.
7:12	76	120	" " Ship rolling much.
7:13	84	110	Standing in cool breeze.
	92	110	" " "
7:15	84	105	
7:29	68	130	Lying after cold tub.
	68	130	"
	68	130	" Cool breeze from port-hole.
	64	125	"
7:35	64	130	"
7:36	80	120	Standing.
	84	95	"
	72	110	"
	76	110	"
7:40	84	105	"
10:06	68	90	" in cool cabin.
	76	95	"
	80	105	" Breeze from port-hole.
	88	100	"
	84	105	"
10:12	76	100	"
10:13	68	120	Lying. Fulness in head.
	68	120	"
	64	115	" Occipital headache.
	72	120	" Weather cold. Dark day.
	72	130	" Feels chilly.
	72	120	" Sleepy. Disinclined to get up.
	68	120	"
	76	120	"
	68	120	"
	72	120	"
	72	120	"
	72	120	"
	68	125	"
	72	120	"

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	72	120	Lying.
	72	120	“
	76	120	“
	76	125	“
	72	125	“
10:45	72	120	“
10:46	80	110	Standing. Headache better.
	84	110	“
	80	100	“ Some fulness in head still.
	84	100	“
	80	105	“ Slight headache.
	84	100	“
10:52	80	100	“ Feels chilly.
p.m.			
12:35	76	90	“ Occipital pain and tenderness.
	76	90	“ Eyes heavy, weak and aching.
	80	90	“ Paræsthesiæ of scalp.
	80	95	“ Fulness in mastoid areas.
12:39	80	95	“ Forehead hot. Hands cold.
12:51	72	95	“
	76	100	
12:53	68	125	Lying. Drowsy. Fulness in head.
	64	125	“ Face flushed. Feet cold.
	64	125	“
	60	125	“
	60	125	“
	60	125	“
	64	120	“
1:01	68	120	“ Headache. Face flushed.
1:02	68	125	“
1:04	68	120	“
1:05	72	115	Standing. Headache better.
	76	90	“ Some fulness in head.
	76	90	“ Eyes ache and feel heavy.
	76	90	“ Absorption and motility tests reacted in 2 hours 16 min.
1:09	72	90	“
6:48	76	105	“ After dinner. Feels well.
	84	105	“
	84	105	“
	84	105	“
	88	110	“
6:56	84	110	“
6:57	80	130	Lying. Fulness in head, especially at vertex.
	80	130	“ Face flushed.
	76	130	“ Pupils normal.
	76	125	“
7:04	76	125	“
7:05	88	120	Standing. Fulness in ears.
	84	100	“

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	76	105	Standing. Frontal headache.
	84	100	" Dizzy. Lump-sensation in stomach.
	88	110	"
	84	110	"
	88	110	"
7:17	84	110	"
10:10	72	105	" After walking.
	76	105	" Feels well. Skin in a glow.
10:13	76	105	"
10:14	76	125	Lying in bed.
	72	130	"
	68	125	"
10:17	68	130	"

Slept well. Port-hole closed because of rough weather.

Protoocol 9.—On "B." Fifth day out on "Caledonia." June 30, 1909.

a.m.			
6:50	64	125	Lying in bed. Feels well. Slight coryza.
	68	120	" Face and hands slightly eczematous.
	68	120	"
	68	125	"
7:00	68	125	"
7:01	80	120	Standing. Arteries very small.
	72	100	"
	76	110	"
	76	110	"
7:05	76	110	"
7:16	76	110	" 2 min. after cold tub.
	76	110	"
	76	105	"
7:20	76	110	"
7:21	68	125	Lying. Slight fulness in head.
	68	125	"
	64	125	"
7:25	64	125	"
7:26	76	125	Standing. Ship rolling considerably.
	76	115	"
	76	110	"
	80	120	"
	72	120	"
7:31	76	110	"
9:48	80	105	" Slight headache.
	88	100	"
	84	100	"
	80	100	"
9:58	80	100	"

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
9:59	72	120	Lying. Feels sleepy.
	72	120	"
	72	120	"
10:02	68	120	" Ship rolling and pitching.
	84	105	Standing. Slight headache.
	84	115	" Feels heavy and dull.
	80	100	" Lightness in head.
	84	100	"
10:07	84	105	"
p.m.			
1:14	76	95	" Before dinner. Arteries small.
	80	105	"
1:16	72	95	"
2:06	80	100	" After dinner. Feels well.
2:07	80	105	" Absorption test reacted in 3 hours 24 minutes.
5:55	64	95	" Feels well, but has indigestion.
	72	105	" Lump-sensation.
	68	105	"
5:58	68	105	"
10:05	76	95	" Slight headache. Lump-sensation.
	72	105	" Fulness in ears made worse by moving head.
	72	95	"
	68	95	"
	72	100	"
	68	95	"
	68	100	"
	68	95	"
	68	95	" Lightness and fulness in head.
	68	90	"
	72	100	" Feels as if top were being raised off head.
	76	95	" Indigestion.
10:21	72	100	" Eyes aching.
10:22	64	120	Lying. Saliva increased.
	56	115	" Lump-sensation in throat.
	64	115	" Fulness in ears, especially in right.
	60	115	"
10:26	60	115	" Ship rolling and pitching.
10:30	60	115	"
10:31	60	115	" Ship rolling mainly.
10:32	60	115	Standing. Lump-sensation comes and goes.
	68	105	" Tinnitus aurium.
	72	105	" Burning sensation in stomach.
	72	105	"
	68	95	"
	72	95	" Lump-sensation in throat.
	76	100	"
10:40	68	105	" Stomach irrigated.

July 1, 1909.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
12:15	72	110	Standing. Feels well.
	72	100	“ Burning sensation in stomach still
	72	100	
12:18	76	100	“
12:19	64	120	Lying in bed. Lump-sensation in throat.
	56	120	“ “
	60	120	“ “
12:22	64	120	“ “

Protocol 10.—On “B.” Sixth day out on “Caledonia.”
July 1, 1909.

a.m.			
8:33	76	120	Lying in bed.
	72	120	“ “
	72	120	“ “
8:36	68	120	“ “
8:37	88	110	Standing. Ship rolling about badly.
	96	90	“ Feels weak.
	96	90	“
	88	90	“ Respiration irregular and rapid.
	88	85	“ Lightness in head.
	92	95	“ Arteries large.
	96	90	“ Fulness in ears, especially behind right mastoid.
	96	90	“
	92	95	“
	104	105	“ Exercised. Chinned bar 3 times.
	104	95	“ Conscious of respiratory movements.
8:49	84	95	“ Muscles weak and tremulous.
9:10	92	90	“ After shaving. Ship rolling badly.
	96	90	“ Has to hold on to berth.
	92	85	“ Stomach feels well.
	92	95	“ Sense of weight in head.
	92	90	“ Eructations.
	92	90	“ Lump-sensation in stomach.
	92	85	“ Saliva free. Numbness in occiput.
	88	95	“
	88	95	“ Feels weak.
9:21	88	95	“
9:22	76	130	Lying. Arteries moderately contracted.
	72	135	“ Respirations 14 to minute.
	72	135	“ Fulness in head.
	72	125	“ Slight lump-sensation in stomach.
	68	125	“ Headache in right temple.
	68	125	“ Face flushed.
	68	120	“ Conjuntivæ congested.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.	
	68	125	Lying.	Saliva free.
	76	120	"	
	72	125	"	
	68	125	"	
	72	120	"	
	68	125	"	
	72	120	"	Respirations 18 to minute.
9:48	72	125	"	Lump-sensation in stomach.
9:49	88	100	"	Eructations.
	84	95	"	Lightness in head behind right ear.
	88	95	"	Numbness behind right ear.
	88	95	"	Respirations 16 to minute.
	88	95	"	Ship rolling badly.
	92	95	"	Feels fairly well.
10:04	88	90	"	Respirations 24 to the minute.
p.m.				
12:50	72	130	"	After making analysis.
	72	130	"	Fulness in head. Skin moist.
	76	130	"	Sleepy. Respirations 16.
	72	125	"	Lightness in left ear.
12:58	72	125	"	Slight headache. Weight on top of head.
12:59	88	110	Standing.	Feels dizzy and weak.
	88	90	"	Tremulousness all over body.
	88	95	"	Fulness and lightness in ears.
	92	95	"	Coronal headache.
	92	90	"	
1:04	92	95	"	
4:40	84	105	"	Feels weak. Sweating.
	84	100	"	Eyes heavy and aching.
	84	105	"	Lump-sensation in stomach.
	84	100	"	
	88	95	"	
4:48	88	100	"	Ship rolling badly.
4:49	68	115	Lying.	Fulness in head.
	72	120	"	Face flushed.
	68	120	"	Sleepy.
	64	120	"	Great relief to lie down.
	68	115	"	Eyes heavy.
	68	115	"	Respirations 22.
	68	115	"	
	64	120	"	
	60	120	"	
	64	120	"	
	64	120	"	
	64	115	"	
	72	110	"	
	68	115	"	
	64	115	"	
	68	115	"	

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	68	105	Lying.
	72	115	“
	68	100	“
	72	115	“
	68	115	“
	72	115	“
	72	105	“
	72	115	“
	72	110	“
	64	115	“
	68	115	“
5:54	68	115	“
5:55	80	125	Standing. Feels well.
	80	105	“ Slight headache.
	80	110	“ Ship rolling badly.
	80	110	“ Numbness and burning in occiput.
	84	90	“
	88	95	“
	84	95	“
6:20	..	..	Dinner over. Absorption test reacted in 37 minutes.
9:34	84	95	Standing after walking on deck.
	84	90	“ Irritable and worrisome.
	76	100	“
	80	100	“
	84	100	“
	84	105	“
	80	105	“
	84	100	“ Feels well.
9:45	84	100	“
9:50	72	120	Lying in bed. Slight frontal headache.
	72	120	“ Pain through right eye.
	68	120	“ Pain in muscles of occiput, right side.
	68	115	“
9:55	68	120	“ Ship rolling badly.

Protocol 11.—On “B.” Seventh day out on “Caledonia.” July 2, 1909.

a.m.			
6:47	72	125	Lying in bed. Feels well.
	76	125	“
	68	120	“
6:54	68	125	“
6:55	..	110	Standing. Lightness in head.
	88	95	“ Feels well.
	72	90	“
	84	90	“

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
6:59	84	105	Standing.
7:00	..	..	Cold tub.
7:27	84	95	Standing. Longs to sit or lie down.
	96	90	"
	92	90	"
7:30	88	85	"
7:31	84	120	Lying.
	72	130	" Throat tickled. Cough suppressed.
	68	120	"
7:34	67	120	"
8:00	68	130	" Slight headache.
	72	130	" Eyes heavy. Sleepy.
	68	130	"
8:04	68	130	"
8:05	68	110	Standing. Numbness in scalp.
	76	100	" Ship rolling heavily.
	76	95	" Feels well.
	76	90	" Conscious of respiratory movements.
	80	95	"
	80	95	"
8:11	88	95	"
8:56	80	100	" Ship rolling. Balancing efforts.
	84	95	"
	76	95	"
8:59	80	95	"
p.m.			
12:20	80	100	" After 3 hours on deck walking and sitting in cool air.
	76	100	" Feels well.
	76	100	" Artery contracted.
	80	100	"
	84	100	"
12:26	80	100	"
12:27	80	120	Lying. Arteries moderately dilated.
	72	120	" Respirations 18.
	72	125	" Feels well. Sleepy.
	68	120	"
	72	125	"
	64	120	"
12:35	64	125	"
12:36	68	100	Standing. Arteries well contracted.
	80	100	"
	80	105	"
	76	100	"
12:40	76	100	"
12:46	76	100	" After effort at balancing.
	88	105	" With eyes closed.
	92	90	"
	84	95	" Arteries dilated.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
12:51	92	95	Standing.
2:52	88	130	Lying. Suppressed a sneeze.
	76	125	" Arteries moderately large.
	76	120	" Respirations 22 at first, later 22.
	76	125	"
	72	120	"
	72	115	"
	76	125	
3:01	72	120	
3:02	76	95	Standing. Feels well.
3:03	92	90	" Arteries large.
3:04	92	90	"
	84	100	"
	92	95	"
	88	95	"
3:08	92	95	"
4:30	80	105	" After sitting on deck for an hour and a half.
	80	105	"
	76	105	"
	80	105	" Arteries small.
	80	105	"
4:35	80	105	"
4:36	84	130	Lying. Face flushed.
	72	130	" Hands cold.
	68	125	"
	68	125	" Feels well.
	72	125	"
	68	130	
4:45	68	130	"
4:46	72	105	Standing. Arteries very small.
	84	100	"
	84	105	"
	80	100	"
	80	105	"
	84	100	"
4:52	84	105	"
			After dinner absorption test did not react positively for 3½ hours.
10:35	88	85	Standing. Feels well.
	88	85	" Arteries very small.
	80	85	"
	92	85	"
	80	85	"
	80	85	"
	80	85	"
	80	85	"
10:45	76	85	"
10:46	60	130	Lying. Arteries large.
	60	125	" Liver not enlarged or tender.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	60	120	Lying.
10:49	64	120	Slight lump-sensation in stomach. Slept fairly well, but dreamed much.

Protocol 11a.—On "S." Aboard "Caledonia." Seventh day out. July 2, 1909.

p.m.			
4:57	84	80	Standing.
	88	90	"
	80	90	"
	84	85	"
	84	100	"
	80	80	"
	80	75	"
	84	80	"
5:22	84	80	"
5:23	80	115	Lying. Arteries large.
	64	105	" Pulse-rhythm irregular.
	56	110	" Face flushed.
	60	100	"
	52	105	" Sleepy.
	56	105	"
	60	105	"
	52	105	"
	56	110	"
	56	105	"
5:35	56	105	"
5:36	56	75	Standing.
	100	80	"
	88	80	"
5:42	100	80	"

Protocol 12.—On "B." Eighth day out on "Caledonia." July 3, 1909.

a.m.			
6:55	68	120	Lying in bed. Slight lump-sensation in stomach.
	72	120	Lying. Respirations 18 and 16.
	72	115	"
	68	115	"
7:01	68	115	"
7:02	88	110	Standing. Lump-sensation in stomach.
	84	100	"
	80	95	"
7:05	80	95	"
7:22	88	85	" After dressing.
	92	90	"

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.	
	88	90	Standing.	
7:27	88	90	"	
7:28	68	120	Lying.	Respirations 20.
	68	115	"	Lump-sensation in stomach slight.
	68	115	"	
	68	120	"	
	68	120	"	
7:37	72	115	"	
7:38	84	85	Standing.	
	84	85	"	
	84	85	"	
	88	90	"	
7:42	84	95	"	
7:51	88	90	"	After stomach irrigation.
	84	90	"	Lump-sensation persists in stomach.
	88	95	"	
	92	95	"	
7:55	92	95	"	
7:56	76	125	Lying.	Fulness in ears.
	80	125	"	Lump-sensation in stomach persists.
	72	125	"	Arteries large.
	68	125	"	Respirations 20.
	72	120	"	
	72	115	"	
	68	115	"	
8:09	68	115	"	
8:10	92	95	Standing.	Respirations 16.
	88	90	"	Raw feeling in fundus of stomach.
	92	90	"	Arteries large.
	88	90	"	
	88	95	"	
8:15	84	90	"	
11:58	68	85	"	After sitting on deck in cool air.
	84	85	"	
	80	90	"	
	76	90	"	
p.m.				
12:05	80	90	"	
12:06	68	115	Lying.	
	64	105	"	
	68	115	"	
	60	110	"	
	60	115	"	
12:10	84	105	"	
12:11	72	90	Standing.	Arteries small.
	76	90	"	
	76	95	"	
12:14	76	90	"	

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
1:38	..	..	Dinner over. Motility test reacted in 1 hour 25 minutes.
1:50	92	95	Standing. Fulness in ears.
	96	59	"
	92	95	"
	88	95	"
	96	90	"
1:56	92	95	"
1:57	76	125	Lying. Respirations 20.
	76	125	"
	76	120	"
	80	115	"
	76	120	"
	76	120	"
	72	115	"
2:31	72	120	" Asleep.
	80	115	" " Respirations 18.
	80	115	" " Pupils contracted.
	76	115	" "
2:35	76	115	" " Conjunctivæ congested.
	72	115	" "
	72	120	" "
	80	125	" " Coughed.
	76	115	" "
	80	115	" "
2:43	76	115	" "
	72	115	" "
	72	115	" "
2:50	68	115	" "
2:51	80	125	" Awoke suddenly.
2:52	80	115	" Hot flush along spine on awaking.
2:53	84	85	Standing. Ship rolling.
	92	95	" Efforts at balancing.
	84	95	" Feels well.
	92	95	" Lump-sensation in stomach.
	88	95	"
	96	85	"
	96	85	"
	96	90	"
3:01	88	90	"
4:57	80	90	" After sitting in cold air.
	88	100	"
	84	90	"
5:05	84	90	"
5:06	76	120	Lying. Face flushed.
	72	120	"
	68	120	"
	76	115	"
5:10	72	120	"
5:11	80	110	Standing. Feels well.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	80	100	Standing. Slight lump-sensation in stomach.
	84	95	"
	84	95	"
	88	95	"
	88	90	"
	88	95	"
5:18	80	90	"
5:35	..	..	Stomach contents removed. Some fresh blood.
10:22	80	90	Standing. After resting on deck.
	84	90	"
	84	85	"
	80	85	"
	84	85	"
10:28	84	90	"
10:29	68	115	Lying in bed. Feels well. Arteries large.
	64	115	" Respirations 20.
	64	125	" Slight lump-sensation in stomach.
	60	120	" Slight *herpes labialis* appearing.
	60	125	"
	60	115	"
10:35	64	120	"

Protocol 12a.—On "S." Eighth day out on "Caledonia." July 3, 1909.

p.m.			
12:19	76	80	Standing.
	72	85	"
	76	85	"
	76	85	"
	76	90	"
	72	95	" Laughed.
12:25	76	85	"
12:34	60	110	Lying. Arteries moderately large.
	60	105	"
	60	105	"
	56	105	"
12:38	60	105	"
12:39	76	85	Standing. Arteries moderately contracted.
	76	90	"
	76	90	"
	76	90	"
12:43	76	95	"

Protocol 13.—On "B." Ninth day out on "Caledonia." July 4, 1909

a.m.			
6:45	..	..	Cold tub. Moville.
7:04	64	125	Lying. Ship has just started from Moville.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	64	120	Lying.
	64	120	"
7:06	68	120	"
7:07	84	105	Standing. Feels well.
	88	105	"
	80	105	"
	84	105	"
7:11	80	105	"
7:54	72	120	Lying. After packing suit-cases.
	76	120	"
	68	120	"
7:57	72	120	"
7:58	96	95	Standing. For a moment blood-pressure was at 115.
	92	90	Standing.
	92	90	"
	92	95	"
8:02	92	95	"
9:40	112	85	" Cabin very hot and close.
	112	90	" Sweating.
	108	90	" Lightness in head.
	108	90	"
	108	80	" Arteries variable, i.e., small at times and at others large.
9:49	104	85	"
9:50	76	135	Lying. Headache. Fulness in head.
	80	125	" Respirations 24.
	80	120	" Feels well.
	76	115	"
	80	120	" Pupils normal.
	80	115	"
	84	115	"
10:01	84	115	"
10:02	112	90	Standing. Arteries moderately large.
	112	85	" Conscious of respiratory movements.
	108	90	"
	108	85	"
	104	90	"
	108	90	"
	108	100	"
10:09	104	90	"
11:25	88	105	" After walking in cold air (57° F.).
	84	95	" Feels well.
	88	95	"
	88	85	"
11:29	88	90	"
11:30	84	105	Lying. Arteries larger than when standing.
	76	105	"
11:32	72	105	" Respirations 20, 22.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
11:33	76	115	Lying.
	68	115	“
11:37	76	115	“
11:38	84	100	Standing. Feels well.
	84	95	“ Arteries moderately dilated.
	84	90	“
	80	85	“
	92	80	“
	84	85	“
	84	90	“
11:45	84	95	“
p.m.			
12:15	100	85	“ Feels well.
	92	85	“ Arteries moderately large.
	92	85	“
12:19	96	85	“
12:20	92	125	Lying. Arteries large.
	80	120	“ Respirations 24.
	80	115	“
	80	110	“ Arteries smaller.
	76	115	“ Fulness in head. Headache.
12:28	80	120	
12:29	76	85	Standing.
	92	80	“
	88	85	“
	96	85	“
	96	85	“
	92	85	“
12:35	92	85	“
12:47	84	..	Standing on deck in cold air.
12:48	96	..	“
12:49	92	..	
12:50	92	..	
12:51	80	..	
3:33	72	95	After standing on deck until feet grew tired.
	72	95	Standing.
	72	90	“
	76	95	“ Arteries small.
	72	90	“
	84	90	“
	80	85	“
3:40	76	95	“
3:41	76	115	Lying. Feels well. Arteries small.
	72	115	“
	72	110	“
3:46	68	110	“
3:47	84	95	Standing. Just before landing.
	80	90	“
	84	95	“

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	84	90	Standing.
3:51	76	95	“
3:52	..	..	Landed at Stobcross Quay, Glasgow.
4:38	96	105	Standing in hotel room, Glasgow.
4:39	88	105	“
4:40	92	115	Lying.
	88	115	“
4:41	84	120	“
4:42	92	110	Standing.
	88	105	“
4:47	84	105	“
	76	120	Lying.
	72	115	“
	72	115	“
	68	115	“
4:50	68	110	“
	68	110	“
4:53	68	115	“
4:54	80	95	Standing. Feels well.
	80	95	“
	88	100	“
	84	100	“
	84	110	“
4:59	84	100	“
9:46	92	95	“ After stroll in botanical gardens.
	84	95	“ Arteries moderately contracted.
	84	100	“
	96	105	“
9:53	88	95	“
9:54	68	110	Lying in bed.
	68	110	“
	64	115	“
9:57	68	115	“

Protocol 14.—On “B.” July 5th, on shore.

a.m.			
8:25	60	100	Lying in bed after awaking.
	64	100	“ Arteries contracted. Room cool.
	64	100	“
8:28	68	100	“
8:29	64	110	“ After turning from head to foot of bed.
	64	110	“
	68	110	“
	64	110	“
	64	115	“
	64	110	“
8:43	64	110	“

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
8:44	88	105	Standing. Feels well. Arteries contracted.
	88	110	“
	80	110	“
	88	110	“
	84	110	“
8:49	84	110	“
9:15	84	100	“
	88	110	“ After dressing.
	88	110	“ Arteries contracted.
	88	100	“
	84	105	“
9:22	80	105	“
9:23	64	110	Lying. Feels well. Arteries contracted.
	64	115	“
	64	115	“ Respirations 20.
	64	115	“
9:29	60	115	“
9:30	84	105	Standing. Arteries contracted.
	84	105	“
	80	105	“
	80	105	“
9:34	80	105	“
p.m.			
5:55	92	120	“ Before leaving for train to catch Dublin boat.
	92	125	“
	96	125	“
5:58	92	125	“
5:59	80	125	Lying.
	76	125	“ Respirations 24.
	72	125	“
	72	125	“
6:07	72	120	“
6:08	80	125	Standing. Arteries moderately dilated.
	88	125	“
6:10	92	120	“
	92	115	“
6:12	88	110	“

Protocol 14a.—On “B,” aboard the steamship “Tiger” of Duke's line from Glasgow to Dublin. July 5, 1909.

p.m.			
7:58	108	95	Standing in cabin. Sweating after exertions.
	108	90	“ Cabin hot.
	104	95	“ Temporary lump in stomach.
	108	100	“
	108	95	“
8:02	..	..	Boat starts.
8:03	108	95	Standing.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
8:05	108	95	Standing.
8:06	92	120	Lying. Slight lump-sensation in stomach.
	84	120	" Arteries smaller but still large.
	84	115	" Fulness in head. Respirations 26.
	84	120	" Slight pain and numbness in occiput on right side.
	84	115	
	88	115	" Boat jolts and trembles.
	84	115	
	84	110	
	80	105	" Jarring of body from head to foot.
	80	105	" No rolling or pitching.
	80	105	" Arteries large.
	76	105	" Respirations 24.
	80	105	" Worrying about trifles.
	80	105	" Deep respirations.
	84	115	" Lump-sensation in throat.
	80	110	" Respirations 24.
8:32	80	115	" Arteries moderately contracted.
8:33	96	85	Standing. Arteries small.
	104	90	" Slight lump-sensation in stomach.
	96	95	" Biparietal headache.
	96	90	" Sweating.
	96	95	" Fulness in ears and mastoid areas.
	96	90	"
	92	95	"
	96	90	"
	100	95	" Headache occipital, right side.
	100	90	"
	100	85	" Lightness in head.
	100	85	"
	96	85	" Lump-sensation marked.
	96	90	"
	100	85	" Pupils slightly dilated.
	100	85	"
	100	85	" Muscles feel in fair condition.
	100	85	"
	96	85	" Numbness in occiput.
	96	85	" Boat pitching somewhat.
	100	85	" Much jolting from machinery.
	92	90	"
	96	85	" Slight pain near umbilicus.
	96	85	" Occipital numbness and aching worse on right side.
	96	90	"
	100	90	"
	100	90	"
9:05	100	90	" Arteries moderately contracted.

July 6, 1909.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
12:10	76	95	Standing. After walking on deck.
	72	95	" Occipital headache worse on left side.
	72	100	"
	76	95	"
	72	95	"
12:15	64	95	" Arteries moderately contracted.
12:16	64	120	Lying. Respirations 24.
	68	125	" Arteries moderately contracted.
	68	125	" Coronal headache.
	68	125	" Face flushed.
	64	125	" Pupils moderately dilated.
	60	125	" Frontal headache.
	68	125	" Respirations 21. Headache. Lump-sensation in stomach.
6:20	68	110	Lying in bed after awaking.
	68	110	" Arteries moderately large.
	72	110	" Respirations 19.
	72	110	" Yawning
	72	115	"
	68	115	"
6:30	68	115	"
6:31	88	105	Standing. Arteries moderately contracted.
	84	95	"
	84	95	"
	88	100	" Arteries moderately contracted.
	80	95	"
	84	100	"
	88	100	"
	84	95	" Arteries small.
	84	95	" Feels well.
6:41	88	95	
7:00	..	..	Landed at the North Wall, Dublin.

Protocol 15a.—On "B," aboard the "Lady Wolseley" of the British and Irish Steam Packet Company from Dublin to Southampton. July 10, 1909.

p.m.			
4:54	..	..	Boat started.
4:55	104	95	Standing. Arteries large.
	100	95	
	108	90	
	108	90	
	104	95	
5:03	104	95	Standing.

Time. p.m.	Pulse-rate.	Blood-press.	Remarks.
5:04	92	120	Lying. Boat steady, but much vibration from machinery.
	96	120	"
	96	115	" Arteries large.
	92	115	" Pupils moderately dilated.
	92	115	
	96	115	
	88	115	
	92	105	Lying. Asleep.
	92	105	" " Respirations 22.
5:15	92	110	"
5:16	112	95	Standing. Arteries moderately large.
	104	85	" Feels well.
	100	95	
	104	95	
	100	90	
	104	95	
5:21	104	90	
6:32	84	95	Standing. After being on deck.
	88	95	" Arteries moderately contracted.
	84	105	"
	92	100	" Weather calm.
6:36	92	105	"
7:16	96	100	" After dinner.
	92	105	
	96	105	
	92	105	
	92	100	
7:21	92	105	
9:55	96	95	Standing. After walking on deck.
	96	95	
	96	95	" Fresh breeze.
	100	95	
	88	95	" Boat pitching a little.
	96	90	" Lump-sensation and burning in stomach.
	92	95	
	92	90	" Boat rolling considerably.
	96	90	
	92	90	" Feeling sick at stomach.
	92	85	
	88	85	" Saliva increased.
	92	90	
	92	85	
10:15	92	90	
10:16	80	135	Lying. Great relief to lie down.
	80	135	" Arteries large.
	76	135	" Lump-sensation in stomach.
10:20	80	135	" Respirations 24. Sleepy. Very nervous.

Protocol 15b.—July 11, 1909.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
5:55	80	135	Lying in bed. Awoke at 4 A.M. after a stormy night.
	76	130	" Boat rolled and pitched furiously.
	76	130	" Marked "lump-sensation" in stomach as boat rolls.
	76	130	" Feels dizzy and sick, though lying in bed.
	72	135	" right side out with head toward bow.
	76	135	" Pain in occiput right side.
6:15	76	135	"
6:16	84	100	Standing.
	84	100	"
6:18	88	100	"
6:20	92	110	" Arteries contracted.
	88	105	" Ship rolling badly.
	88	115	" Marked balancing efforts.
	92	105	" Lump-sensation in stomach.
	80	110	" Saliva increased.
6:27	80	115	" Heaviness in head.
6:28	80	135	Lying. Lump-sensation very marked.
	80	135	" Mind dull. Sick headache.
	76	135	" Saliva much increased.
	72	135	" Occipital headache.
	72	135	" Pain in neck.
	76	125	" Arteries moderately contracted.
6:34	76	130	
7:15	..	..	Stomach irrigated. Mucus and some fresh blood removed.
8:05	80	125	Lying. After test meal.
	80	125	" Sick, heavy feeling in head.
	80	125	" Dull coronal headache.
	80	130	" Saliva free.
	80	135	" Lump-sensation in stomach.
	80	135	" Respirations 22.
	80	135	" Arteries very small.
	80	135	
	80	135	" Sleepy. Face flushed.
	80	135	Occipital headache.
	80	135	Lying. Disinclined for work.
	80	135	" Irritable. Wants to be let alone.
	76	135	
8:24	80	130	" Ship rolling badly.
8:25	86	125	Standing. Marked efforts at balancing.
	92	110	" Nervous. Sweating.
	88	115	"
	88	115	" Feels weak.
8:29	88	110	"
11:00	88	125	" Ship rolling badly.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	92	120	Standing. Much effort at balancing.
	96	110	" Arteries contracted.
	92	110	" Feels weak from balancing.
11:04	84	105	
p.m.			
1:50	88	105	" After dinner. Boat steady now.
	96	105	" Arteries moderately contracted.
	92	105	
	88	115	" Whiskey and soda at dinner.
	80	105	"
1:55	92	105	"
3:25	..	..	Alongside pier at Falmouth.
4:40	100	105	Standing after making analyses.
	92	105	" Boat at pier.
4:43	92	105	"
9:43	76	110	" After walking on deck.
	80	110	" Respirations 24.
	80	110	" Arteries moderately contracted.
	80	105	"
	80	105	" Feels well.
9:50	80	110	"
9:51	76	125	Lying in bed. Boat still by Falmouth pier.
	72	125	" Arteries moderately large.
	68	125	"
	72	125	" Respirations 20.
	68	125	"
10:05	72	125	" Feels well.

Protocol 15c.—July 12, 1909.

a.m.			
7:45	76	125	Lying in bed after awaking.
	72	125	" Boat still at Falmouth pier.
	72	120	" Arteries moderately contracted.
	72	120	"
	72	120	" Respirations 22.
	72	125	"
	72	120	"
8:03	72	120	"
8:04	100	105	Standing. Arteries moderately dilated.
	100	110	" Feels well.
	96	105	"
	96	105	"
	92	105	"
	88	110	"
	92	105	"
9:09	96	105	"
9:10	80	140	Lying after effort of getting into berth.
	72	135	" Arteries moderately large.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	72	125	Lying.
	76	125	"
9:18	80	125	" Boat started for Plymouth at 9:17 A.M.
9:19	104	95	Standing. Arteries moderately large.
	100	95	" Feels well.
	96	105	"
	96	105	"
	96	100	"
	92	105	"
9:25	96	100	"
10:49	92	105	" Boat moving smoothly.
	88	105	"
	92	105	" Feels well.
	88	105	"
	92	105	"
10:55	92	105	"
10:56	72	140	Lying. Arteries moderately contracted.
	72	140	" Respirations 18, 20.
	72	140	" Pupils normal.
	76	140	" Sleepy.
	76	135	" Slight "lump-sensation."
	76	135	
	76	135	" Much jolting from machinery.
	76	135	
11:11	80	135	" Occipital headache.
11:12	100	105	Standing. Arteries moderately contracted.
	88	105	"
	88	105	" Slight occipital headache.
	84	105	"
	88	110	"
	88	105	"
	92	105	"
	88	105	"
	84	105	"
11:21	88	105	"
p.m.			
3:00	..	..	Arrived at Plymouth. Strolled about town.
6:00	..	..	Boat started for Southampton.
7:14	92	110	Standing after dinner.
	96	110	"
	100	105	"
7:17	96	105	"
7:18	76	120	Lying. Arteries moderately large.
	88	125	"
	88	125	" Respirations 20.
	88	120	"
	88	120	"
	84	120	"
7:25	84	120	"

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.	
7:26	108	95	Standing.	Face flushed.
	100	105	"	Fulness in head.
	96	105	"	
	96	110	"	Feels stupid and heavy.
	100	105	"	
7:31	96	105	"	
10:42	76	100	"	After a bottle of ale.
	80	105	"	Slight dizziness.
	80	105	"	
	80	105	"	Fulness in head.
	80	105	"	
	76	105	"	
	88	105	"	
	80	105	"	
	84	100	"	
10:58	80	105	"	
10:59	76	125	Lying in bed.	Fulness in head.
	72	125	"	Face flushed.
	72	120	"	
	76	120	"	
	72	120	"	
11:09	72	120	"	Slept poorly.

Protocol 15d.—July 13, 1909.

a.m.				
5:43	72	125	Lying	after awaking.
	72	125	"	Slight lump-sensation in stomach.
	72	120	"	Respirations 24.
	76	125	"	
	72	120	"	
	72	120	"	
	76	120	"	
5:56	72	120	"	
5:57	104	95	Standing.	Gliding up "Southampton Water."
	96	95	"	Arteries contracted.
	92	95	"	
	88	95	"	Feels sick at the stomach.
	88	95	"	Sweating.
	88	105		
	88	100	"	Feels weak.
	92	110	"	
	92	105	"	
	96	95	"	
	96	100	"	
	88	95	"	
6:10	88	100	"	
6:12	..	..	Landed at Southampton.	

Protocool 16a.—On "B," aboard the "Southwestern" from Southampton to Cherbourg. July 13, 1909.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.	
11:50	96	110	Standing.	After walking from the Hippodrome, i.e., about ¼ of a mile.
	92	110	"	
	88	120	"	Cabin stuffy.
	88	115	"	Arteries moderately contracted.
	92	105	"	
	92	105	"	
	96	105	"	
			July 14, 1909.	
	88	105	Standing.	
	92	110	"	
a.m.				
12:01	88	105	"	
12:02	76	125	Lying.	Respirations 24.
	76	125	"	Twitching of long flexors in right wrist.
	76	130	"	
	76	130	"	
12:06	76	130	"	
12:11	76	130	"	
12:12	96	110	Standing.	Arteries small.
	92	115	"	Feels well.
	92	115	"	
	88	110	"	
	92	105	"	
12:20	88	105	"	
12:24	..	..	Boat started.	
1:37	92	95	Standing.	Arteries moderately dilated.
	88	105	"	Feels well.
	88	105	"	
	88	105	"	
	88	105	"	
1:43	88	105	"	
1:44	76	125	Lying in bed.	
	76	130	"	Arteries moderately contracted.
	72	125	"	Feels well.
	72	125		
	68	125		
1:51	72	125		
6:33	76	125	Lying, after awaking.	
	76	125	"	Boat rolling and pitching.
	76	120	"	Awoke with lump-sensation in stomach.
	80	120	"	

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	76	120	Lying. Saliva free.
	80	120	
	80	120	
6:50	80	120	“ Feels nervous.
6:51	80	115	Standing. Some balancing efforts.
	92	120	“ Marked lump-sensation.
	96	115	
	96	110	“ Feels weak.
	96	120	“
	96	110	“ Saliva increased.
	92	105	“ “ “
	96	115	
	96	105	“ Sweating.
	92	110	
	92	105	“ Arteries much contracted.
	92	110	
	92	110	
7:20	..	..	Landed at Cherbourg.

Protocol 16b.—On “B,” aboard a train. July, 1909.

a.m.			
9:22	92	125	Sitting in train before starting.
	96	125	“
9:25	88	125	“
9:26	88	120	“ On moving train.
	88	95	“
	92	120	“
9:29	88	110	“
9:30	88	115	“ At a stop.
9:31	88	105	“ On rapidly moving train.
9:32	88	105	“
9:33	88	115	“ At a stop. Arteries very small.
	88	120	“
9:35	88	120	
9:36	88	115	“ At a stop.
	84	120	“
	80	120	
9:40	80	120	“
9:41	80	125	Lying. At a stop.
9:42	68	125	

Protocol 17.—On “B,” after dressing ashore. July 19, 1909.

a.m.			
6:07	84	105	Standing after dressing ashore.
	80	105	“ Feels well.
	80	105	“ Arteries small.
6:12	80	105	“
6:13	68	125	Lying. Arteries larger.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	68	125	Lying.
	68	125	“
6:16	64	125	“
6:17	76	115	Standing.
	80	115	“
	80	120	“
6:20	84	115	“
			July 20, 1909.
7:15	..	..	Cup of tea.
7:30	84	105	Standing after dressing ashore.
	88	115	“ Arteries contracted.
	88	130	“
	88	130	“
	88	130	“
7:41	88	130	“
7:42	80	135	Lying. Arteries larger than when standing.
	72	140	“
	72	135	“
7:45	72	140	“
7:46	88	125	Standing. Arteries contracted.
	88	130	“
	88	125	“
7:49	88	125	“
7:50	..	..	Boarded steamship *Cygne* bound for Gorey, Island of Jersey, Channel Islands, from Carteret, Manche.
8:15	..	..	Boat started.
8:16	..	..	Ewald breakfast. Absorption test reacted in 15 minutes. Motility test reacted in 1 hour 20 minutes.
8:24	88	130	Sitting. On deck in hot sun.
	88	125	“ Boat rolling and pitching.
	88	130	“ Lump-sensation in stomach.
	84	120	
8:32	88	135	“ Arteries contracted.
8:33	92	135	“ In the sun.
	84	135	“ Much balancing effort.
	84	135	“ Ship rolling and pitching.
	84	135	“ Feels well in spite of heat.
8:41	96	135	“ Strong odour from cook's galley.
8:42	96	140	“ On deck as before.
	88	130	“ Arteries moderately contracted.
	88	130	“ Slight lump-sensation in stomach.
	88	130	
	84	130	“ Eructations.
9:00	88	130	
9:06	84	130	“ On deck in sun.
	80	135	“ Slight headache.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	80	140	Sitting. Otherwise feels well.
	88	140	
9:10	84	140	"
9:33	84	135	" On deck.
	92	135	" Arteries moderately contracted.
	96	120	"
	96	120	"
	92	125	"
9:43	92	120	"
9:45	..	..	Landed at Gorey.

Protocol 18—On "B," aboard the fishing smack "Quatre Frères." July 22, 1909.

a.m.			
8:53	..	..	Boat started, a fresh breeze filling her sails.
8:54	76	120	Lying in cabin. Feels sick. Arteries small. Odour horrible.
9:00	80	125	" Boat pitching badly. Sweating.
	80	115	" Rapidly getting sicker.
9:22	76	105	" Arteries not so small now.
	76	105	" Sweating. Lump-sensation. Saliva free.
	76	115	" Lump in throat. Wretched all over.
	72	115	" Bathed in cold sweat.
	72	125	" Marked lump-sensation in stomach.
	72	125	" On verge of vomiting. Pain in epigastrium.
	72	115	" Feels better now.
	72	115	" Arteries small. Face pale.
	72	90	" On verge of vomiting again.
	76	115	" Feels better. Sweating.
9:59	72	115	
10:18	72	120	" Arteries small. Feels thoroughly wretched.
12:00	..	..	The rough weather compelled the crew to put back into the harbour. Put ashore.
p.m.			
12:24	80	130	Sitting after leaving boat.
	84	120	" Arteries contracted.
	84	120	"
	80	115	"
	76	115	"
12:27	84	120	"
12:28	76	130	Lying. Arteries moderately contracted.
	80	135	"
	76	135	"
	72	135	"
	76	135	"
12:33	76	135	"

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
4:55	92	125	Standing. Muscles of forearm twitching.
	92	120	" Arteries moderately large.
	96	120	" Feels nervous and irritable.
	92	120	" Lump-sensation still in stomach.
	96	120	"
5:02	92	120	"
5:03	76	125	Lying. Size of arteries varies slightly from time to time.
	68	135	" Pupils normal.
	72	130	" No special fulness in head.
	68	130	"
	76	125	"
5:11	72	130	"
5:12	92	105	Standing. Arteries moderately contracted at times, and at other times small.
	96	105	Standing.
	92	135	"
	92	135	" Lump-sensation still in stomach.
	92	130	"
	92	120	"
	92	135	"
	92	125	"
5:20	88	130	"
			The subject suffered the whole afternoon from the effects of the sea trip.

Protocol 19.—On "B," aboard the "Southwestern," from Cherbourg to Southampton. July 23, 1909.

p.m.			
11:25	80	120	Standing in cabin.
	80	130	" Arteries moderately contracted.
	80	125	"
11:30	80	125	"
11:31	72	135	Lying. Arteries moderately large.
	68	140	"
	68	135	"
11:36	68	135	"
11:37	76	130	Standing. Arteries contracted.
	80	120	"
	80	115	
11:40	84	115	"
11:47	..	..	Strychnin nitrate, gr 1/40.
	..	..	Atropin sulphate, gr 1/75, both hypodermically.

July 24, 1909.

a.m.			
12:04	..	..	Boat started.
1:43	76	125	Lying. Arteries moderately contracted.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	80	135	Lying.
	72	130	"
1:46	80	135	"
			Following an Ewald meal given at 12:34 A.M., the absorption test reacted in 11 minutes. The motility test did not react within the hour.
			The blood-pressures could not be taken with the subject standing on account of the excessive incessant pitching and tossing of the boat.
7:49	76	135	Standing as boat approaches pier at Southampton.

July 28, 1909.

7:50	84	120	Standing. After good night's rest in cool room.
	88	125	" Arteries moderately contracted.
	84	125	"
	84	125	"
	88	125	"
	88	125	"
	84	120	"
	84	120	"
	84	125	"
	84	125	"
8:11	84	120	"
8:12	72	135	Lying. Arteries moderately contracted.
	76	130	"
	76	130	"
	72	125	"
	72	130	"
8:18	72	130	"
8:19	80	130	Standing. Arteries much contracted.
	84	130	"
	84	125	"
8:23	84	130	"

In an Ewald breakfast given at 7:18 A.M., the absorption test reacted in 21 minutes. The motility test did not react within the hour.

Protocol 20.—On "B," aboard the "Teutonic" from Southampton to New York. First day out. July 28, 1909.

a.m.			
11:52	88	110	Standing in cabin.
	88	105	"
	92	110	"

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	92	110	Standing in cabin.
	88	115	"
	96	120	"
	88	110	"
p.m.			
12:04	88	110	"
12:05	80	135	Lying. Arteries moderately large.
	80	130	"
	76	125	"
	76	125	"
	76	125	"
12:13	80	120	"
12:14	100	120	Standing. Arteries moderately contracted.
	92	115	"
	92	110	"
12:17	88	105	" Boat sailed from Southampton at 12:15 P.M.
5:05	80	140	Standing. Arteries small.
	88	135	"
	84	135	"
	84	135	"
	76	135	"
	80	135	"
	84	130	"
5:17	84	135	"
5:18	68	145	Lying. Arteries moderately contracted.
	72	140	" Fulness in ears.
	72	145	"
	68	145	"
	68	140	"
5:25	68	145	"
5:26	76	135	Standing. Arteries contracted.
	80	130	" Room cool.
	80	135	"
	76	130	"
	80	135	"
5:31	80	130	"
7:12	84	150	" After dinner.
	80	145	"
	84	135	"
	88	130	"
	84	140	"
	84	120	"
	88	125	"
7:26	84	115	"
7:27	72	140	Lying. Arteries moderately large.
	72	140	" Slight fulness in head.
	72	140	" Feels well.
	68	140	"
	72	140	"

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	80	135	Lying.
	76	140	"
7:36	80	135	"
7:37	92	115	Standing. Some balancing efforts.
	84	120	" Arteries variable. At times small, at others moderately large.
	84	130	"
	84	105	"
	84	125	"
	84	120	"
	84	115	"
	84	110	"
	88	115	"
	88	130	"
	84	110	"
7:55	84	110	" Feels well.
11:56	80	120	" Arteries moderately contracted.
	80	125	"
	76	120	"
a.m.			
12:01	80	120	"
12:02	72	130	Lying in bed. Arteries slightly larger.
	68	130	"
	68	130	"
12:05	72	130	" Feels well. Boat moved steadily so far. Weather fair.

Protocol 21.—On "B," aboard "Teutonic." Second day out. July 29, 1909.

a.m.			
7:48	68	120	Lying in bed after awaking.
	76	125	" Slept fairly well.
	68	125	" Arteries moderately large.
	68	115	"
	72	120	"
8:04	72	120	"
8:05	88	120	Standing. Arteries moderately small.
	80	115	" Ship moving steadily.
	88	115	"
	88	120	"
	84	115	"
	84	110	" After lying some hours in Queenstown harbour the boat started for New York at 1:10 P.M.
p.m.			
1:33	76	110	Standing. Arteries contracted.
	72	115	" Cabin cold.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	80	110	Standing. Does not feel so well.
1:35	80	110	"
1:36	68	115	Lying. Arteries considerably contracted.
	68	120	" Feels chilly and not so well.
	72	120	"
	68	115	"
1:43	80	110	"
1:44	76	110	Standing. Arteries vary from moderately small to moderately large.
	76	105	"
	76	110	"
	72	115	"
	72	115	"
1:51	80	115	"
6:53	84	115	" Arteries vary, being in general moderately large.
	84	110	"
	88	110	"
6:59	84	105	"
7:00	72	130	Lying. Arteries moderately large.
	72	130	"
	72	130	"
	72	130	"
7:06	68	130	"
7:07	88	110	Standing. Arteries vary considerably.
	88	100	"
	84	100	"
	84	100	"
7:12	84	105	"
10:06	72	105	" Ship pitching.
	76	110	" Arteries vary.
	76	110	" Arteries small.
	76	110	"
	76	105	"
10:12	76	105	"
10:18	68	120	Lying in bed. Arteries moderately large.
	68	120	"
	68	120	"
	68	120	"
	64	120	"
	68	115	"
10:19	68	115	"

Protocol 22.—On "B," aboard "Teutonic." Third day out. July 30, 1909.

a.m.			
7:15	72	115	Lying in bed after awaking.
	72	115	" Arteries moderately contracted.
	72	115	"

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	72	115	Lying.
	72	115	"
7:23	72	115	"
7:24	76	115	Standing. Some balancing efforts. Rough weather.
	84	115	" Ship rolling and pitching much.
	76	125	"
	84	120	"
	84	115	" Condition of arteries variable.
7:29	84	115	"
8:10	80	115	" Arteries variable.
	80	110	"
	88	115	" Feels well.
	88	115	"
	88	110	"
8:15	88	115	"
p.m.			
3:30	84	125	" Balancing efforts.
	84	115	" Ship pitching and rolling badly.
	84	110	" Arteries somewhat contracted.
3:36	88	120	
3:37	72	130	Lying. Arteries large.
	68	125	" Arterial wall quickly recedes from finger.
	68	130	"
3:42	72	130	"
3:43	80	115	Standing. Arteries moderately contracted.
	84	110	" Some balancing efforts.
	84	110	" Ship rolling and pitching.
	84	105	
3:47	88	110	" Cabin warm.
5:00	72	130	" Much balancing.
	80	120	" Very rough seas.
	76	130	"
	76	130	"
5:05	76	130	" Arteries moderately contracted.
9:54	72	120	" Arteries vary slightly from time to time.
	72	115	"
	72	115	"
	72	115	"
	72	115	"
10:02	72	115	"
10:03	64	115	Lying in bed. Arteries moderately large.
	64	115	" Fulness in ears.
	60	115	" Face flushed.
	64	110	"
	64	120	"
	64	110	"
	64	120	"

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	64	115	Lying. Ready to drop into sleep.
10:15	68	115	"

Protocool 23.—On "B," aboard "Teutonic." Fourth day out. July 31, 1909.

a.m.			
7:19	68	120	Lying in bed after awaking.
	68	120	" Slight occipital headache the result of studying future plans.
	68	120	"
	68	120	"
7:26	68	115	Standing. Cabin warm.
	88	110	" Arteries variable.
	92	105	" Sweating.
	88	105	"
	92	105	" Ship steadier.
7:34	92	105	"
11:42	88	105	"
	76	115	" Arteries vary within moderate limits.
	80	115	"
	76	115	" Feels well.
	76	110	"
	76	115	" Occasionally slight lump-sensation in stomach.
	72	115	"
	72	120	"
	72	115	"
11:53	76	120	"
p.m.			
5:14	76	125	Lying in bed after sleeping.
	72	125	" Arteries moderately contracted though slightly variable.
	72	125	"
	72	125	"
	72	130	"
	72	125	"
5:24	68	125	"
5:25	100	115	Standing. Arteries variable.
	92	115	"
	96	110	"
	92	110	"
	88	115	" Cabin warm.
	88	105	" Ship very steady.
	88	110	
5:35	88	105	
10:42	72	115	" Arteries variable, but in general moderately large.
	76	115	"

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	96	105	Standing.
	92	110	"
	88	115	" Ship steady.
	84	115	"
	76	110	" Port-holes open.
	76	110	"
	80	110	"
10:54	76	110	"
10:55	64	120	Lying in bed. Arteries moderately contracted.
	64	120	"
	64	120	"
	64	120	"
	64	120	"
11:02	64	120	"

Protoocol 24.—On "B," aboard "Teutonic." Fifth day out. August 1, 1909.

a.m.			
7:32	64	115	Lying in bed after awaking.
	68	120	"
	68	125	"
	72	125	"
	68	125	"
7:42	68	120	"
7:43	88	100	Standing. Feels well.
	88	105	" Cabin oppressively hot.
	92	100	" Sweating.
	92	105	"
	92	100	"
7:50	92	100	"
8:30	92	100	" After dressing.
	92	105	" No lump-sensation.
	88	100	"
	92	100	" Ship steady.
	92	100	" Arteries large.
	92	100	"
	96	100	" Weather very warm.
8:39	96	100	"
p.m.			
1:30	84	100	" Arteries variable.
	96	100	" Blood-pressure at times lower than recorded.
	92	100	"
	88	105	"
	88	100	"
1:38	92	100	"
1:39	68	135	Lying. Arteries large.

Time, p.m.	Pulse-rate.	Blood-press.	Remarks.
	72	135	Lying.
	68	135	"
1:43	68	145	"
1:44	88	105	Standing. Arteries vary moderately.
	88	105	"
	88	110	"
1:47	88	105	"
4:04	72	120	" After being on deck in cool air.
	76	120	"
	76	120	"
4:08	76	115	"
4:09	64	125	Lying. Arteries moderately large.
	64	125	"
	68	125	"
4:15	64	120	"
4:16	72	115	Standing. Arteries vary slightly.
	80	115	"
	72	115	"
4:20	76	115	"
9:15	88	100	" In hot cabin.
	84	105	"
	84	105	" Arteries variable but for the most part dilated.
	84	110	"
	84	110	"
	84	110	"
	84	105	"
9:23	84	100	"
9:24	68	125	Lying in bed. Arteries moderately large.
	68	125	"
	68	125	"
9:29	68	125	"

Protocol 25.—On "B," aboard "Teutonic." Sixth day out. August 2, 1909.

a.m.			
7:33	64	115	Lying in bed after awaking.
	68	115	" Had a fair night, but the cabin was fearfully hot.
	72	120	"
	68	115	"
	68	115	" Weather very warm.
7:40	68	115	"
7:41	96	100	Standing. Arteries variable, but in general moderately large.
	96	105	"
	92	105	"
	92	100	"

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	84	100	Standing. Sweating.
	92	100	"
	88	100	" Temperature of air in cabin, 90° F.
7:49	92	100	"
11:36	72	120	" In cool cabin. Port-hole open.
	64	130	" Feels well.
	64	125	"
	68	125	"
	68	130	"
11:43	64	130	"
11:44	72	120	Lying. Arteries moderately large.
	64	125	"
	64	125	"
	64	125	"
	60	120	"
11:52	60	120	Standing. Arteries moderately small.
	76	120	"
	72	120	"
	72	125	"
	72	120	"
11:58	72	125	"
p.m.			
1:05	56	130	Lying after sleeping.
	60	125	"
	64	125	" Arteries moderately contracted.
	64	125	"
	64	125	"
1:11	60	125	"
1:12	72	130	Standing. Arteries vary, but in general are contracted.
	72	130	"
	68	125	"
	72	130	"
	68	130	"
1:17	68	135	"
9:38	72	110	" Arteries moderately large.
	72	115	"
	72	115	" Temperature of air in cabin, 68° F.
	76	115	"
	72	115	"
9:45	76	115	"
9:46	68	120	Lying in bed. Arteries moderately large.
	68	125	"
	64	125	"
	64	125	"
	64	125	"
	64	120	"
9:53	60	120	"

Protocol 26.—On "B," aboard "Teutonic." Seventh day out. August 3, 1909.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
7:37	64	125	Lying in bed after awaking.
	64	125	" Slight headache from thinking over future plans.
	64	125	"
	68	125	"
	64	120	"
7:46	64	120	"
7:47	80	110	Standing. Arteries variable.
	84	110	" Temperature of air in cabin, 74° F.
	84	110	"
	80	100	"
	80	100	"
	80	100	"
	80	110	"
	84	115	"
	88	115	"
7:56	88	115	"
p.m.			
5:40	84	110	" Arteries variable.
	80	115	" Sweating.
	80	115	" Temperature of air in cabin, 81° F.
5:43	80	110	" Cabin stuffy.
5:44	68	130	Lying. Arteries moderately large.
	68	135	" Tired after walking on deck.
	64	130	"
5:47	64	125	"
5:48	76	115	Standing. Arteries variable.
	80	115	"
	84	110	"
5:52	80	110	"
11:36	72	120	" Arteries moderately contracted.
	72	120	"
	72	115	"
11:40	72	115	"
11:41	64	120	Lying in bed. Arteries moderately contracted.
	64	125	" Delightful cool night.
	64	125	"
11:45	64	125	"

August 4, 1909. Eighth day out on "Teutonic."

a.m.			
7:52	64	115	Lying in bed after awaking.
	68	120	" Arteries moderately contracted.

Time, a.m.	Pulse-rate.	Blood-press.	Remarks.
	68	120	Lying. Temperature of air in cabin, 69° F.
7:56	72	120	"
7:57	92	105	Standing. Arteries moderately contracted.
	84	110	"
	88	115	"
8:01	84	115	"

August 5, 1909. Boat lying in New York Harbour.

a.m.			
5:41	68	120	Lying in bed after awaking.
	68	120	"
	68	120	"
5:46	68	120	"
5:47	88	115	Standing in stuffy cabin.
	88	115	" Temperature in cabin, 71° F.
	88	115	"
5:50	88	115	" Arteries moderately contracted.
8:00	..	..	Landed in New York.

CHAPTER XXX

HISTORY AND LITERATURE OF SEASICKNESS

Judging from the extent of its literature, seasickness has been a much discussed question in the past. All sorts of theories have been adduced to explain the causation of its phenomena. Many of the articles written by the older men contain ingenious theories backed with numerous clinical facts and sustained by vigorous argument.

Hippocrates alludes to the perturbations of the body caused by navigation. Cicero was a victim and it was even said of him that sooner than endure the seasickness he suffered on the vessel in which he had taken refuge from Mark Antony he gave himself up to his executioners.

Pliny [276] has a good word for some drink as a remedy: "Nauseam maris arcet in navigationibus potum."

Cato [229] the Elder, said that one of the greatest regrets of his life was that he had made a journey by sea that he could have made by land. Lord Bacon was also a sufferer from seasickness and carried a sachet filled with saffron as a preventative.

Many other names holding high place in the history of science, art, and letters, might be mentioned to show what little regard the sea has for the condition or personality of those committed to its tender mercies.

Erasmus Darwin [230] (1796) attributes the vertigo from circumgyration (vertigo rotatoria) to an increase of the irritative motions of vision and when the rotatory motions are continued too long or are too violent, sickness of the stomach follows. To a similar cause he attributes the sickness following vinous intoxication and the motions of a ship. Wollaston [231] (1810) considers the inertia of the blood to be the cause of seasickness. As the boat descends a pressure equal to that of a six foot column of blood is taken off the blood-vessels so that they contract and cause congestion of the brain.

Jobert[232] (1846) cites the theories of his time, and attributes seasickness to indigestion caused by disturbances of the sympathetic nervous system.

Pellarin[233] (1851) considers the cause as a disturbance of the circulation, the symptoms being due to cerebral anæmia.

Sewanas[234] wrote a large volume in which he contended that seasickness was due to a miasmatic poison existing in the atmosphere over the sea. The miasma originated in the decomposition of organic detritus in the sea, and was therefore more abundantly manifest when the sea was agitated.

Guillabert[235] (1859) accepted the theory of Sewanas as demonstrated.

Berard[236] (1850) attributes the vomiting of seasickness to disturbance of the central nervous system.

Nelken[237] (1856) observes that few persons are immune. Children under 2 years are exempt, but children from 2 to 7 years are more liable, whilst after 7 the susceptibility is the same as in adults. He states that dogs, sheep, cows and chickens are subject to the malady, whilst hogs, ducks and geese are unaffected. His view is that the motions of the ship are communicated to the various viscera within the abdomen, thorax, cranium and spinal canal, and that according to the idiosyncrasy of each individual various symptoms manifest themselves.

Chapman[238] (1864) considered spinal congestion as the cause of seasickness, and strongly recommended a specially constructed ice-bag applied to the whole length of the spine. Many good results were reported. The travellers were instructed however to lie flat upon the ice-bag, i.e., in dorsal decubitus.

Barker[239] (1870) considers seasickness as due to the sudden and recurring changes of the relations of the fluids to the solids of the body, and to the nervous disturbances which result from these changes.

Pollard[240] (1872) attributes seasickness to the consciousness of a want of support in the downward movements of the ship, causing thereby strong psychic irritation.

Guien[241] (1876) divides the causes into those which affect the nervous system as a whole, such as the motions of the boat, and those which affect particular nerves as bad odours, noises, etc.

Beard[242] (1881) attributes seasickness to a series of concussions. He denies that the symptoms of seasickness are "reflexed through the eyes." Infancy and old age escape; neurotic individuals are more susceptible.

Kramer[243] (1892) studied the fundus oculi in seasickness, and found in most of the cases the "vessels thin and narrow and the chorioid of a pallor otherwise found after protracted hemorrhages." He considers cerebral anemia as the cause of seasickness, the psychic element being an important factor. The cause of the cerebral anæmia he leaves an open question.

D'Ailhaud Castelet[244] (1895) attributes seasickness to abnormal excitement of the sensory nerves of the skin and viscera and to irritations of the vagus, sympathetic, sense organs, muscular sense and of the sense of space which induces peripheral vaso-constriction with compensatory visceral vaso-dilatation, hypnosthenia of the heart muscle, lowered blood-pressure, cerebral anæmia and miosis.

Tousey[245] (1896) considers most cases of seasickness as due to reflex causes from the visual apparatus or from the semicircular canals. Few cases are caused by succussion of the stomach.

Rosenbach[246] (1896) repudiates fear, abnormal visual impressions, deficient function of the kidneys or lungs and miasmastic poisoning, as being at best merely associated factors and not the cause of seasickness. The movements of the ship are the all-important factor and of these the screw or spiral motion is the most effective. The stamping or pitching motion is next in effect, whilst the rolling motion is the least effective of all. He also states that it is at the "end of the upward motion" and the "beginning of the rise" that the greatest influence is exerted, i.e., when the direction of the motions is reversed. All authors, he says, agree on this point. These movements according to Rosenbach affect the body by causing intra- and inter-molecular disturbances of dynamic equilibrium within the organism. He believes that vomiting is due to a definite mechanical influence upon the organic tissue of the stomach. The vomiting is a functional interference without the primary intervention of a nerve influence.

Hazen[247] (1904) attributes seasickness to a deficiency in innervation of the extrinsic muscles of the eye. On a vessel we have the severest test for "immunity of the panoramic symptom." The condition for prevention of seasickness is to have the standard strength of these muscles.

Klein[248] (1897) attributes seasickness to the effect of pressure, traction and concussion upon the nervous elements in the movable organs.

DeVries (1899) considers seasickness as a functional disease of the nervous system, caused by repeated mechanical agitation of the body.

Darnall[250] (1899) concludes that seasickness results from a disturbance of equilibration caused by disturbance of the endolymph in the semicircular canals. He makes no attempt to explain the *modus operandi.*

Sumner[251] (1900) relates the story of a house which had been carried away in a flood, and on the subsidence of the waters left in a tilted position, partly in the water and partly on dry land. No part of the house floated or moved. Every one who entered this house experienced feelings like those of seasickness, and some even vomited. He considers seasickness as due to a "flood of conflicting impressions conveyed to the mind through vision," and, therefore, as purely a psychic disturbance.

Schwerdt[252] (1901) attributes seasickness to circulatory disturbances, with disordered equilibrium as a cause of secondary importance. He bases his conclusions upon manometric measurements of intra-abdominal pressure, registered by "an ordinary manometer which was alternately connected with the stomach and the bowel (rectum) by suitable insertions." Although it is now known that such measurements of pressure, taken within the hollow viscera, by no means represent intra-abdominal pressure, yet the author repeated Schwerdt's experiments and found no such constancy in the phenomena as reported. Great credit is, nevertheless, due to Schwerdt as being one of the first to approach the study of seasickness in accordance with the scientific spirit of the times. It is a relief to find some one in the literature checking the monotony of a mere expression of views by appeal to experimental fact.

Savory[253] (1901) considers the cause as purely reflex and physiological, acting primarily through the semicircular canals. He considers the connection between the upper division of the auditory nerve and the geniculate ganglion, and between the latter and the vagus, as forming a complete chain from the auditory to the pneumogastric terminals. Savory is the only one so far who has made any attempt to trace the paths of nerve impulses, or to commit himself fearlessly to an open declaration of his views. It is now known that no pathway exists between the vagus and the eighth nerves within the ear. Savory is entitled to credit for having made the first attempt at a rational explanation of the phenomena of seasickness.

Weitlauer[249] (1903) after numerous observations and measurements, concludes that seasickness is due to traumatism originating in repeated differences in pressure of the fluid and soft elements of the body, as compared with the solid elements.

Cornelius[255] (1903) seeks the cause of seasickness in a combination of Rosenbach's kinetic theory with his own pressure-point theory, the nerve points being irritated by the disturbances of equilibrium, thereby sending stimulating impulses to the centre.

Binz[256] (1903–4) considers seasickness as due to cerebral anæmia caused by stimulation (from the rocking motions) of the vaso-motors of the brain. He, however, admits the possible connection between cerebral ischæmia and involvement of the organs of equilibration. Binz used Kreidl's onychoscope, an instrument which registers on a dial plate the rate and strength of the pulse in a nail segment, in his investigations, and found that under rhythmical lowering and raising of the arm, the vessels became filled in a notably slower manner than they emptied themselves. The plethora is permanently overcompensated by a relative ischæmia during the vibrations.

Pfanz[46] (1903), from his observations with the aid of Kreidl's onychoscope, Basche's sphygmomanometer, and Gaertner's tonometer, held it as probable that an alternate inflow and outflow of the blood in the brain induced by the rocking motions of the boat which raise and lower the head, causes cerebral stimulation, which in turn is responsible for the retching and vomiting of seasickness.

He also observed an increase in blood-pressure at the same time. Both Binz and Pfanz are entitled to credit for applying experimental methods to the study of seasickness.

Hagen-Torn[105] (1903) considers seasickness as due to cerebral anæmia, caused by reflex contraction of the cerebral vessels. "The impossibility of adaptation to the constantly changing relations of the body toward its surroundings" is the primary cause of the phenomena.

Koepke[259] (1904) believes that seasickness is due to cerebral anæmia.

Waugh[260] (1904) considers the cause of seasickness to be a paralysis of the vaso-motor nerves with accumulation of blood in the viscera (abdominal) and anæmia elsewhere, especially in the brain.

Shelmerdine[261] (1904) insists that a predisposition is necessary in order that the ship's motion may cause nausea. Seasickness results from a combination of three factors, viz.: (1) mechanical, i.e., overwork of the muscles in balancing; (2) mental; and (3) irritation from the presence of bile in the stomach.

Corning[262] (1904), by means of a revolving chair induced vertigo, nausea, and cardiac weakness. Then he experimented with various drugs with a view to finding a remedy which would hold the phenomena in abeyance. He found a combination of hyoscin and morphin to be the most effective. Observing the similarity between the phenomena of rotation sickness and those of seasickness he next proceeded to treat seasickness in the light of his findings. On a voyage he himself took hyoscin hydrobromid, gr 1/50; opium, gr ss; and ten minutes later resorcin, gr iii, with nitroglycerin, gr 1/300. The result was wonderful. He seemed to be in such a state that he could not be made sick, and went to the bow of the ship in defiance of the elements. He treated twenty other passengers, to whom he gave a combination of morphin, atropin, cocain, and resorcin, and found that "thus torpid at centre and periphery, the subject usually remained proof against giddiness and nausea for from four to seven hours." A tablet was then given containing morphin, gr 1/6; nitroglycerin, gr 1/300; strychnin sulphate, gr 1/60; resorcin, gr i; cocain hydrochlorid, gr

1/6. This was sufficient to procure further immunity for a like period. Most of his subjects, however, preferred to lie down after taking the drugs. Corning does not attempt to explain the causation of the phenomena of rotation sickness or of seasickness. He deserves credit for making a practical attempt to find a remedy for seasickness by invoking the aid of experimental methods and applying the results of his findings to practice.

Thoma[263] (1904) attributes seasickness to a "deficient sense of equilibrium," this faculty being imperfectly developed in the majority of individuals. He states that seasickness is never observed among birds, in which the organ of equilibrium is most highly developed, and acrobats. Children or infants, and those suffering from incurable disease, are usually spared.

Zingher[264] (1905) considers the etiology of seasickness as still obscure, and does not commit himself to an opinion. He eulogizes a proprietary preparation in the treatment of seasickness.

Regnault[265] (1906) distinguishes between psychic and somatic seasickness. Bonnet, Berillon, Van Renterghem, and Farez hold similar views.

Roesen (1907) and Simon (1907) evidently consider cerebral anæmia to be the cause of seasickness. They employed Bier's method of congestion for the relief of the condition. Each believes he saw good results from this method.

Simon[254] used an adjustable elastic necktie, which was applied to the neck for half an hour at a time.

Flasschoen[284] (1907) attributes seasickness to a dynamic disturbance at the base of the brain, and especially in the optic thalamus and corpora striata. He adduces no facts or reasons for his assumption.

Barnett[285] (1907) attributes seasickness to irritation of the auditory nerve terminals by movements of the endolymph in the semicircular canals. This irritation is transmitted to the vagus through the facial. Possibly, also, the irritation is transmitted to the sympathetic system through the external petrosal branch of the geniculate ganglion. It is now known that the apparent connection between the facial and auditory nerves (fila anastomica) consists merely of aberrant strands of facial fibres that return to the facial after temporary association with the auditory.

Maillet[257] (1908) considers seasickness as the result of reflexes from all the floating organs.

Van Trostenburg[258] (1908) considers seasickness as the result of disorientation in space associated with a series of interacting disturbances arising from unaccustomed optic, kinæsthetic, and labyrinthine irritations.

Various other theories have been advanced from time to time, most of which fall under one or other of these heads, as given by Rosenbach:

1. The localization theory.
2. The somatic (mechanical) theory, which includes: (a) the theory of circulatory disturbances; (b) centrifugal force theory; (c) the cerebral theory; (d) the abdominal theory; (e) theory of the static centre.
3. The theory of psychic and ocular vertigo (the influence of will-power, fear, etc.).
4. The theory of intra- and inter-molecular disturbances (Rosenbach's theory).

CHAPTER XXXI

ETIOLOGY OF SEASICKNESS

Of the various theories put forward to account for the phenomena of seasickness not one affords a reasonable explanation that is applicable to any large number of cases. An exception, perhaps, must be made in the case of those theories which attribute the malady to disturbances of the endolymph in the semicircular canals (Barnett, Savory, and others). Even here no proof has been offered to show that such disturbances are in fact the cause of seasickness; and with the exception of attempting to show a continuity of fibre-paths (now known not to exist) between the auditory nerve and the pneumogastric through the facial (Savory, Barnett), no effort has been made to explain the *modus operandi*, or to outline the mechanisms and structures involved.

Before attempting to establish the cause of seasickness, a few words are necessary about the psychic factor so often mentioned in connection with the malady. In a certain limited class of persons the psychic element is undoubtedly of importance. Thus in a previous chapter it was stated that one of the subjects of these studies became ill from merely boarding his ship on the eve of commencing a journey. All the indications afforded him good reasons for believing that he would become dreadfully seasick on the voyage. Here was a case where evidently the psychic element was paramount. Analysis, however, shows that this of itself is not sufficient proof that psychic causes alone can primarily produce the phenomena of seasickness. On this occasion the underlying factors were subconscious (latent) memories of previous experiences, which themselves were the direct result of genuine seasickness produced in the usual way. Moreover, in this instance the psychic factor had the support of powerful associated causes, viz., the foul odours and the dismal, unsightly aspect of affairs between decks. As we shall see later, psychic, like circulatory and gastric disturbances, are always associated with seasickness to a greater or less extent; and, like them, may be potent, secondary sources of discomfort. This, however, does not elevate them to the dignity of being the cause of seasickness in the strict sense. Many individuals of the neurotic type, whom one would expect to be the victims of seasickness, are immune; whilst, on the other hand, the most robust individual, with every indication of a well-balanced nervous system, may get sick every time he goes to sea. The author has known a woman of remarkably neurotic type who had been the victim of seasickness all her life, until a certain age, when she unexpectedly began to develop immunity. Simultaneously with her immunity there developed bilateral nerve deafness, which was hereditary in her family. This woman's sister had her hearing impaired equally in both ears early in life, and was always immune to seasickness, although, judging from the psychic and neurotic standpoint, every appearance gave indication that she ought to be a victim. In middle life, however, owing to acute disease, she became totally deaf in one ear; and, on the next occasion of her going to sea, she discovered that she had developed a susceptibility for seasickness, and could get relief in no other way than by lying upon the back or upon the side in which the hearing had been destroyed. It will be remembered that the sheep and dog, with unilateral division of the vestibular nerve, lay upon the side of operation, because in any other position they experienced vertigo and developed nystagmus. The immunity of deaf mutes to galvanism over the mastoid areas and to rotation-sickness (James,[266] Kreidl,[267] and others), and of animals in which both labyrinths have been removed (Ewald,[277] Högyes[278]), or both vestibular nerves divided (Schiff[279]), is relevant here.

The onset of mild unilateral inflammation of the middle ear, or even the unilateral exacerbation of old middle-ear disease, may render a subject who had been previously immune very susceptible to seasickness or car-sickness. One such case was recently studied. The patient was a woman of thirty-three, whose hearing had been very poor since the age of four, when she had had severe bilateral otitis media suppurativa, complicating scarlet fever. This patient had not been subject to sea- or car-sickness within her memory, and yet she was of a distinctly neurotic type. In her fifth month of pregnancy she went shopping and found, to her dismay, that riding in the cars not only made her weak, dizzy, and nauseated, but initiated cramps, which strongly stimulated uterine contractions. The patient had no fever, but showed, upon examination, distinct evidence in the left ear of acute exacerbation of the dormant inflammatory conditions existing in both ears. She had had a slight pain in the left ear for two or three days. With this pain she noticed that her hearing for high notes had improved. There was some tenderness to pressure in the left ear. On standing with the eyes closed the patient was inclined to fall toward the right. Turning the head from one side to the other, i.e., rotating it upon the long axis of the body, made her dizzy and sick. With the eyes turned to the left, there was well-marked horizontal and rotary nystagmus to the left and with the watch. With the eyes turned to the right there was a mere trace of rotary movement, which was slow and directed against the watch. With the eyes directed up and down there was no visible nystagmus. The knee- and wrist-jerks were equal on either side of the body and somewhat hyperactive. The pupils were equal, and exhibited the usual rhythmic alternations of short range, with the phase of dilatation always the more salient. Hurrying or moving about rapidly made the patient weak and dizzy, and evoked the abdominal pains referred to. At times the sight was blurred and external objects seemed to move, but the direction of their movements was not observed.

The treatment, which was very effective, included dorsal decubitus, dry cold over the mastoid area of the affected side and bromides. It should be mentioned that the occasion on which the car-sickness manifested itself was a hot, oppressive day in September. Judging from the well-known effect of hot weather upon rotation-sickness and seasickness, it is highly probable that this patient's sickness was aggravated by the humidity and closeness of the day. There were no symptoms referable to disordered gastric, liver, or kidney function; in fact, the woman's physical condition was excellent both before and after the aural attack.

Another case worth recording is that of Mrs. S., at one time a well-known professional dancer. The natural strength and stamina with which nature had endowed this lady was greatly enhanced by the daily training necessitated by her profession. Notwithstanding all this, however, and although accustomed from infancy to all sorts of gyrations and sudden reversals of direction, this individual was a victim of seasickness to such an extent that she never fully recovered from her attacks until she got ashore. At a certain period of her life, the exact age could not be ascertained, her system underwent a change which rendered her immune to seasickness. Thus, her last attack of seasickness occurred on returning from Europe in 1859. Between the years 1859 and 1869 she had not been on the water. In 1869 she began to have trouble with her ears. Impairment of hearing followed immediately, and persisted ever since. In 1890 a relative placed his yacht at her disposal, and it was only after the greatest amount of persuasion that she could be induced to venture aboard for a short trip. To her great surprise she found no discomfort in the roughest seas. Since that time she has travelled extensively upon the water under extremes of weather conditions, and never experienced the slightest symptoms of seasickness. Judging from the experience gathered from numerous cases in which aural irrigations and other methods were practised, it seems certain that the vestibular terminals undergo degeneration at an early age in many individuals, although Witmaack[26] is of the opinion that in toxæmic conditions affecting the auditory nerve, the vestibular branches escape. In the individual under consideration the change which conferred immunity from seasickness undoubtedly was due to degeneration, whereby the receptors in the ampullæ of the semicircular canals, and possibly also those in the maculæ of the utricle and saccule, lost their sensibility so that they no longer responded to the stimulus generated by the movements of the ship.

These cases show that even in very neurotic individuals something more than merely psychic factors is the underlying cause of seasickness. Admitting the important relations between the cerebrum and the cerebellum, and admitting that psychic disturbances always accompany the severer forms of seasickness, and may even act as secondary sources of distress and discomfort, often to such an extent as to seem to be the chief cause of the malady, it must be denied that such a thing as seasickness from purely psychic causes exists in the sense that psychic causes are, in the first instance, the active factors in the production of the malady. Subconscious or conscious memories from past experiences in seasickness, may so affect the individual that very little in the way of direct physical disturbance suffices at times to induce sickness. In such cases perhaps the sickness may be considered as of psychic origin, whereas in reality the cause in the first instance was physical and not psychic. Moreover, in these so-called psychic cases there is always the necessity of some physical cause in addition to the psychic factor; and where the former is wanting, seasickness, somatic or psychic, will not be much in evidence.

It is well known that disturbances of ocular functions can cause nausea and vertigo (Stevens [280] and others). De Cyon [74] put prismatic spectacles upon pigeons and obtained disturbances of equilibrium akin to those which follow interference with the semicircular canals. In pigeons which had partially recovered from the effects of operation upon the semicircular canals, covering the head and eyes with a hood caused a return of the characteristic phenomena (Ewald,[76] De Cyon [74]). The most that these experiments show is that the eye is a factor in equilibration, and this is freely admitted. Ocular defects, such as errors of refraction, diploplia, muscular weakness, anything that interferes with binocular vision and the conjugate action of the eyes may cause disturbances of equilibrium, nausea, vertigo, and localized neurasthenia of the nervous mechanisms involved. The latter may develop a generalized neurasthenia. But all this does not show that ocular disturbances are the cause of seasickness. Admitting that the eye is an important source of afferent impulses in the mechanism of equilibration, and admitting that in certain extreme cases of disturbed function, the eye may be the chief apparent factor in seasickness, coupled perhaps with overmastering psychic impressions, as in the case of Sumner's tilted house, how are we to account for the numerous cases of seasickness occurring in robust individuals with practically perfect eyes and a normal condition of the nervous system and general musculature of the body? Merely because in seasickness the functions of the eye and brain become deranged, and the use of these organs proves a source of discomfort to the individual, are we therefore to conclude that psychic or ocular disturbances, or both combined, are the every-day cause of that seasickness which so few escape when the weather conditions are sufficiently severe? It were as reasonable to say that, because a hood placed over the eyes of a pigeon recovering from the effects of section of the semicircular canals, caused a return of the disturbances of equilibrium, the phenomena first observed by Flourens are therefore due to interference with vision, and not to destruction of the semicircular canals. Finally, not every ocular defect causes vertigo, nausea, and disturbances of equilibrium, whereas the slightest interference with the functions of the ampullary receptors of the semicircular canals, e.g., aural irrigation, rotation, galvanism, the application of cocain, touching, exposure to light, etc., have the immediate and constant effect of overthrowing the equilibrium and causing vertigo, nausea, nystagmus, increased salivation, etc. On the whole, even in extreme cases, the most that can be said of the eyes as a cause of seasickness is that they may become secondary sources of disturbance in the malady. Whenever they seem to play a leading rôle in the causation of seasickness, it is because of associated subconscious impressions or actual disturbances of the oculo-cerebellar or vestibulo-cerebellar mechanisms, and even then it is not certain that the prime cause at work may not be the vestibular rather than the ocular mechanisms. As previously noted, the retinal and labyrinthine receptors and their afferent arcs make use of the same final common paths in the causation of nystagmus. The motor mechanisms of the eyes are, therefore, intimately related to the labyrinthine mechanisms. Hence, in disturbed conditions of the latter there is also associated disturbance of function in the former, and looking at moving objects, such as the tossing waves or fixing the eyes as in reading, becomes very distressing. However, in normal persons, the eye can stand prolonged looking at moving objects, without causing vertigo or sickness, provided the labyrinthine mechanisms are intact and are not interfered with in their functions. As the primary cause of seasickness the eye, and with it the psychic factor, may therefore be excluded.

Concussion of the organs, abdominal or other, has been frequently put forward to explain the causation of seasickness. Undoubtedly, concussion and shaking about can cause serious disturbances in certain individuals. The latter, however, will generally be found to be abnormally constituted in some respect or other, especially if they react abnormally in this manner to concussions of mild degree. Thus in many individuals who suffer from carsickness, recent uncompensated labyrinthine defects may be found, or perhaps a localized or a general neurasthenia. In grown people many cases of carsickness are the result mainly of fear, the cause being originally the individual's experience at some time in youth when, on account of acute ear disease, sickness attended every attempt at riding in cars. These experiences often leave behind them impressions that subconsciously dominate the individual to such an extent that in after years, when the labyrinthine defect has been long since recovered from, or compensated, the slightest excuse in the way of a related physical cause suffices to evoke the disagreeable symptoms. Such cases are frequently encountered by those making special inquiry into the matter. The cases which react to mild concussions, however numerous they may be, are few compared with the number of normal individuals whom such slight concussions as those experienced in riding upon cars do not at all affect disagreeably.

Severe blows upon the abdomen or severe injury to any part of the body may cause disturbances which are grouped clinically under the name of shock. The difference between shock and the condition presented by seasickness, especially in the earlier and milder stages of the latter, is so apparent that no comment on this point is necessary. Blows on the head, especially upon the lower jaw as seen in the prize ring, frequently cause vertigo, vomiting, muscular weakness, etc. In these instances the delicate mechanisms of the labyrinth are the parts chiefly affected, and it is to disturbance of the vestibulo-cerebellar mechanisms that the familiar "grogginess" is mainly to be attributed, as well as the muscular weakness that for the time being renders the contestant helpless, a ready victim for the *coup-de-grâce.* The vast number of those who can withstand concussion of varying degrees of severity on shore, such as that encountered in horseback riding, etc., and yet who are nevertheless susceptible to seasickness from the comparatively mild degree of concussion from the ship's movements, points conclusively to something other than mere mechanical concussion as causing the phenomena of seasickness. This was well shown in those auditory irrigations in which water of a neutral temperature, on being allowed to flow into the auditory canal from a height in order to determine the effect of forcible impact of the fluid against the drum membrane. The only effect was a marked rise in blood-pressure which any strong stimulation at the periphery would have produced. No labyrinthine phenomena were observed, although, when fluids of a temperature sufficient to evoke vestibular phenomena were used, the effect of the irrigations seemed to be enhanced when the fluid was allowed to flow into the ear under pressure. Gross concussion of the body, as a whole, plays a very subordinate part in the etiology of seasickness, though it must be admitted that in the highly disordered state of the individual already suffering from seasickness, concussion vibration, or any other form of peripheral irritation, may be a source of aggravation and distress, and may materially retard recovery.

Disturbances of the circulation have always held a prominent position in the theories advanced to explain seasickness. A glance at the protocols will show that the subject was not seasick during periods when the circulation was but poorly maintained, whilst, when he was sickest, e.g., on board the *Quatre Frères* and the *Lady Wolseley,* the blood-pressure was well maintained. It is admitted that in seasickness after a time the circulation becomes demoralized; but what is sought now is the primary cause and not merely the effects or associated phenomena of seasickness. Moreover, aural irrigations caused characteristic manifestations of vestibular disturbance even when the action of adrenalin was at its height. Rotations also produced the usual vertigo, nausea, etc., when the subject was under the influence of nitroglycerin, with its low blood-pressure, and of morphin, with its well-maintained blood-pressure. As before stated, circulatory disturbances are to be considered as one of the results of seasickness, but they may in turn assume importance as secondary sources of exhaustion and distress.

Digestive disturbances have always been looked upon by the laity as the primary cause of seasickness. But the fact that one of the subjects "B" of the present studies suffered at times from indigestion, and did not at all experience the familiar subjective phenomena of seasickness, whilst at other times he was dreadfully seasick when the stomach, after being irrigated, was presumably in a resting state, points to deeper underlying causes. Even admitting the importance of digestive disturbances in seasickness, the question remains as to what is the cause and what the *modus operandi* of such digestive disturbances. This, however, has to be said of digestive disturbances in seasickness, and the same holds true for the sickness of rotations and aural irrigations, that when once the stomach function becomes disordered the presence of stagnant contents or the ingestion of improper food is a powerful and distressing factor, and remains so until the stomach is emptied by vomiting, when the sickness and distress seem to be relieved for a time. The symptoms, however, especially those referable to the head, recur soon after in spite of the empty condition of the stomach. Here, again, aural irrigations and galvanism by inducing all the stomach phenomena of seasickness with the subject sitting quietly or lying down, dispose forever of those theories which set up concussion of the abdominal organs, whether floating or otherwise, as the sole cause of seasickness. Digestive disturbances are, therefore, to be regarded in the first instance as the effects of seasickness. When once initiated, however, they may become important secondary sources of suffering and distress.

The theory of intra- and inter-molecular disturbances of Rosenbach may or may not be true. Ultimately the cause of seasickness might be stated in such terms, but meanwhile we have a right to know the cruder and more tangible phenomena that intervene. It is a noteworthy fact that, as long as the real cause of an ailment remains undiscovered, theorists deceive themselves with generalizations, which lay down predisposing and subsidiary factors and associated phenomena as the primary efficient cause. This was what happened in the case of tuberculosis before the time of Koch's discovery of the tubercle bacillus, and this may be expected to happen in every case, as long as the real cause remains undetermined. The true cause of such a malady as seasickness should afford a reasonable explanation of the phenomena of most, if not all, of the cases, and of the mode of their production. It should, moreover, be in accord with, and have the positive support of known anatomical and physiological facts. Such a cause in the case of seasickness is to be found in functional labyrinthine disturbances, due directly to the boat's motions, and producing in turn effects in the manner which we shall now endeavor to elucidate.

CHAPTER XXXII

ETIOLOGY OF SEASICKNESS (CONTINUED)

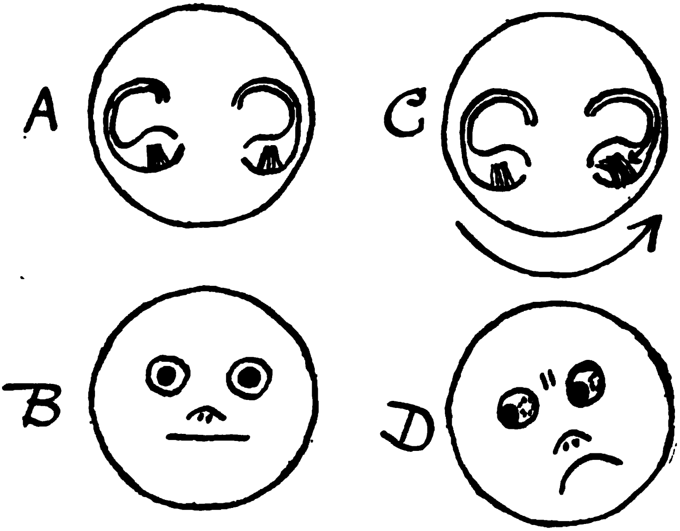
It has been shown that the phenomena attending rotations, aural irrigations and galvanism applied to the mastoid areas are identical with the Flourens phenomena that follow section or destruction of the semicircular canals in animals. Numerous observations on the circulatory and gastric functions, as well as a comparison of the objective and subjective symptoms, show an undeniable similarity between the phenomena of seasickness and those produced by rotations, aural irrigations, and galvanism. The only difference is in the degree and in the mode of production. It has been shown that in aural irrigations and in galvanism the phenomena were caused by a relative difference in the degree of irritability of one set of ampullary receptors, as compared with the conditions of irritability obtaining in the analogous opposing set of ampullary receptors in the opposite labyrinth, and that such a difference in irritability upsets the balance of the reflex vestibulo-cerebellar mechanisms by giving an undue effect to the normal impulses of equilibration originating in the labyrinths, with consequent disturbances of equilibrium, nystagmus, muscular inefficiency, alternating conditions of the pupil, varying conditions of the vaso-motor mechanisms, closure of the pylorus, nausea, vertigo, increased salivation, vomiting, faintness, sweating, pallor, paræsthesiæ, etc. In rotations identical results were produced by actual overstimulation of the terminals of one side, noted especially on sudden stopping and reversing of the swing. The effects of rotations in each of the three cardinal planes of the body were worked out in detail, and constant relations were found to exist between certain sets of canals and rotations in a certain plane. A certain type of nystagmus was found to correspond with rotations in a certain plane, as well as with certain kinds of aural irrigations and with certain applications of the electrodes in galvanism. Thus rotations about the long axis affect the horizontal canals mainly, and cause horizontal nystagmus. Hot and cold aural irrigations and galvanism similarly act mainly upon the horizontal canals, as owing to the location of the latter their ampullary receptors are more exposed to the effects of aural irrigations and galvanism than the receptors in the other canals. When the irrigations are prolonged, or for some reason the effect of the current is enhanced, the ampullary receptors of the adjacent superior canal are affected, with the result that a rotary element is added to the horizontal nystagmus. A similar rotary element is frequently met with in the nystagmus of rotations, e.g., when the rotations about the long axis are not strictly confined to one plane.

In rotations it has been shown that in the case of the horizontal canals the creation of a tendency in the endolymph to flow toward the ampulla from the canal was the physiological stimulus, whilst in the case of the other canals the reverse was true, viz., the creation of a tendency in the endolymph to flow from the ampullæ toward the canals. It was also shown that each canal or set of canals, for in the case of the superior and posterior canals the two superior canals are opposed to the two posterior, was related to certain sets of skeletal and ocular muscles through the intermedium of the cerebellum, and that any stimulation of the ampullary receptors in one set more than in the other was followed by reflex disturbances of equilibrium, and when repeated, by nausea, vomiting, increased salivation, etc. These disturbances have been shown to be chiefly due, not to any simple reflex act involving Deiters' or any other single nucleus, but to reflex acts taking place under cerebellar influence, which extends not only to ocular and skeletal muscles, but perhaps also to visceral, secretory and other functions as well.

The vestibulo-cerebellar mechanisms have been fully discussed as well as their relation to vertigo. It has been shown that interference with the afferent arcs of any of the cerebellar mechanisms, whereby the balance between certain mechanisms upon one side of the body and their opponents upon the other side was upset, caused vertigo and disturbances of equilibrium until such interference was overcome or compensated. The relations of the vagus and glosso-pharyngeal nerves to gastric movements were pointed out, irritation of the vagus causing pyloric closure, whilst irritation of the glosso-pharyngeal tends to cause relaxation of the cardiac sphincter. The relation of these two nerves to each other and to the seventh through their reception nuclei in the sensory portion of the dorsal nucleus, and the manifest association of increased salivation with nausea and vomiting point to the related motor nuclei in the nucleus ambiguus and vicinity, acting in conjunction with some higher centre (vomiting centre) as the co-ordinating mechanism underlying this symptom complex. The relation of the olfactory mechanisms to nausea was pointed out and the pathways indicated along which olfactory impulses, which may be such important secondary features in nausea and seasickness, might travel. The widespread relations of the peripheral sensory nerves to the vaso-motor and vomiting centres were also discussed.

A word remains to be said about vomiting which, in rotation sickness as well as in seasickness, is of the cerebellar type. In experiments upon the semicircular canals in sharks (Sewall[281]) and upon the cerebellum in higher animals (Ferrier[8]) no vomiting was witnessed. In the latter instance, however, as all anæsthetists know and as Ferrier himself has frequently pointed out, profound narcosis or shock tends to prevent vomiting. Sewall found no constant characteristic disturbances of equilibrium following destruction of the semicircular canals in sharks, but found that vomiting frequently occurred upon interference with the utricle or saccule.

The experiments in which cold bilateral aural irrigations were first given to deaden the ampullary receptors of the horizontal canals and rotations then practised immediately, seem to indicate that labyrinthine receptors, other than those located in the semicircular canals, are related to the mechanisms of vomiting. Thus, after the rotations mentioned there was no nystagmus or vertigo, no deviation of the head, or sense of distress, and yet the



DIAGRAMS SHOWING HOW ROTATION OR THE MOVEMENTS OF A SHIP TEND TO CAUSE REFLEX DISPLACEMENT OF THE HEAD AND EYES AND WITH THESE DISTURBANCES OF EQUILIBRIUM VERTIGO, NAUSEA, ETC.

A and *C* represent imaginary transverse horizontal sections through the head and the horizontal semicircular canals. In *A* the subject is at rest. In *C* he is just beginning to be rotated from right to left, as indicated by the large arrow. *B* represents the position of the head and eyes with the semicircular canals and their hair cells in a state of rest, as depicted in *A*. In *C* the hair cells in the left horizontal canal are slightly bent in the direction from the canal toward the ampulla by the tendency of the endolymph to flow in the direction of the small arrow. This causes reflex displacement of the head and eyes toward the right, as shown in *D*. The dotted pupils in *D* indicate the short nystagmic movements to the left.

subject vomited violently. As the rotations were strictly about the long axis, it is inferred that the receptors in the utricle, and perhaps those in the saccule, were affected and that these were directly responsible for the vomiting and alterations in the blood-pressure. Again in rotations given with the patient in recumbency, vomiting was absent, but appeared at once if the subject assumed the erect posture. This seems also to point toward the conclusion just deduced. It seems, therefore, that the otolithic apparatus of the vestibule is dynamic as well as static in function, as pointed out by Clark,[6] and there can be little question that these structures are affected in rotations as well as the ampullary receptors of the semicircular canals. However, the hair cells in the ampullæ are much more delicate than those in the maculæ, and are consequently more susceptible to mild grades of stimulation. Hence, in the milder forms of seasickness it is probable that only the ampullary receptors are affected, whilst in rough weather, where the pitching and tossing of the vessel are unusually severe, the receptors in the utricle and saccule may also be affected.

Considering the chief motions of the ship at sea, they are found to correspond roughly with rotations about one or other of its main axes. Thus rolling may be regarded as rotary movement about the long axis and pitching as rotary movement about the horizontal transverse axis. In the combination of pitching and twisting, known as the spiral movement, an element of rotation about the vertical axis is added. Of the various motions the most effective for producing sickness is the spiral movement. Next in order of efficiency comes pitching. Rolling seems to be the least effective of all. The analogy of these movements to the movements of the subject's body in the rotation experiments is at once apparent. With the subject lying on the back and with the long axis of his body parallel to the long axis of the ship, the rolling movement gives a reproduction in miniature of rotations about the long axis. The comparison may seem far-fetched, but it has been shown that in rotations little effect was experienced when the motion was equal and uniform, even though rapid, whilst great disturbance was produced by the sudden stops and starts. In the rotating motions of a ship, complete revolutions may not be made as in the swing, but the stops and starts are only too painfully in evidence, as every victim of seasickness knows. With the subject still on his back and his long axis parallel to that of the ship, pitching gives a rotatory movement, corresponding to rotations in the sagittal plane. Here, again, the stops and starts are what tend to sicken, though in the pitching of a ship the abruptness of these depends much upon the range of motion. When the twist or spiral movement is added to pitching, the new element is equivalent to rotations in the coronal plane, about a horizontal antero-posterior axis through the umbilicus.

With the subject lying on the back, but with his long axis at right angles to that of the ship, the rolling motion is equivalent to rotation in the sagittal plane, pitching to rotation about the long axis, and when the spiral motion is added, the new element is equivalent to rotation in the coronal plane.

With the subject standing so that the sagittal plane is parallel to the long axis of the ship, rolling is the equivalent of rotations in the coronal plane, and pitching the equivalent of rotations in the sagittal plane, whilst an added twist or spiral movement represents rotations about the long axis. When the subject stands with the sagittal plane at right angles to the ship's long axis, rolling is the equivalent of rotation in the sagittal plane and pitching the equivalent of rotation in the coronal plane, whilst an added spiral movement represents rotation about the long axis.

Each of these movements has been shown to affect definite portions of the semicircular canal apparatus. Thus rotations about the long axis affect the horizontal canal on the side toward which the rotation takes place, by creating a tendency for a current of endolymph to flow from the canal toward the ampulla, causing primarily a displacement of the head toward the shoulder of the opposite side and a primary nystagmus in the same direction as that of the rotation. Sudden stopping of this motion reverses the phenomena with sickening effect, and with a great tendency to muscular exhaustion and rapid alternating changes in the vaso-motor tonus. Rotations in the sagittal and coronal planes cause characteristic nystagmus with displacements and disturbances, the effect depending upon whether the superior or posterior set of canals are affected in the rotations in the sagittal plane, or whether the right or left superior canal is affected in the rotations in the coronal plane. It was found that rotations about the long axis, with the subject sitting upright, were the most efficient in causing sickness, and this corresponds with what is known of the effects of the spiral movements of a ship upon an individual sitting upright or standing. In rotations and irrigations practised with the subject in recumbency the effect was always less than when rotations and irrigations were practised with the subject sitting upright, whilst irrigations or galvanism over the mastoid areas, practised with the subject standing, gave the quickest and sharpest reactions. Here, again, the results of experiment are corroborated by common experience. With the subject in recumbency, rotations when brisk and frequently repeated, and irrigations with very hot or very cold water and frequently repeated, registered their effects upon the central nervous system; and, although the subject was frequently not sufficiently affected as to make him very sick, yet on assuming the erect position, he almost invariably vomited. Once more common experience corroborates experimental findings. Many passengers on the first night at sea go to bed feeling comfortable, and awake in the morning to find, on attempting to stand up, that during the night something had taken place which had paved the way for the extreme distress that culminates in vomiting.

In the upward and downward motions of a ship, rectilinear movement must also be taken into consideration, as such movements, when violent, undoubtedly affect the otolithic apparatus in the utricle and saccule, and enhance the efficiency of the spiral and pitching movements in causing seasickness. In marked downward and upward movements the normal reflexes (other than those mediated through the labyrinth), controlling equilibrium, are also markedly disturbed. The result is a twofold disturbance of equilibrium by (1) distortion of the normal afferent impulses to the cerebellum, and by (2) distortion of the normal impulses to the other centres, controlling gross movements of station and equilibrium, e.g., the optic thalamus, etc. Thus, when a part of the ship, having attained its maximum height in pitching, commences its rapid descent, the afferent kinæsthetic impulses from the proprioceptors in the lower limbs misrepresent the true condition of affairs and the corresponding efferent motor impulses sent out in response, are out of all proportion to the actual needs. The result is a temporary loss of the sense of position of those parts which form the essential basis for cerebellar action in the maintenance of equilibrium. Disturbance of the afferent impulses from the cord to the cerebellar and other centres might, perhaps, be compensated by the semicircular canals, as Bickel[282] has shown by division of the posterior spinal nerve roots in the dog. In seasickness, however, the semicircular canals themselves are so affected that they could scarcely be expected to compensate defects in other mechanisms.

The relations of each semicircular canal or set of canals to definite groups of skeletal and ocular muscles, as well as the relations of the vestibulo-cerebellar mechanisms to other important functions have been fully discussed in previous chapters. The frequent absence of well-marked nystagmus in seasickness is no reason for making a fundamental distinction between seasickness and rotation-sickness. In some persons the nystagmus in rotations is slight and transitory. Similarly in many persons a mild galvanic current will effectually sicken the individual without causing much apparent nystagmus. This is especially true if the current be opened and closed frequently for a long time without rest or intermission. Moreover, even in the milder grades of dizziness or vertigo nystagmus will always be found if searched for with the ophthalmoscope.

The views herein set forth as to the etiology of seasickness receive corroboration from various sources. Thus in elderly persons whose auditory nerves are well known to undergo atrophy and degeneration, there is a comparative immunity from seasickness as well as from the sickness that comes from aural irrigations, etc. Tests have been frequently made upon such individuals with aural irrigations and with galvanism, and it was generally found that they had diminished susceptibility. The immunity of deaf-mutes to rotation sickness, as well as the acquired immunity and susceptibility in the four cases already alluded to, afford direct evidence of the rôle of the vestibular nerve elements in the causation of seasickness.

It should always be remembered that where the labyrinthine receptors are equally affected on both sides simultaneously, there is no apparent disturbance of equilibrium. Thus, in persons who rapidly become almost completely deaf, there may be no vertiginous symptoms. This happened in Witmaack's[26] case, where sudden bilateral deafness, occurring in a young phthisical subject, was caused by toxic acoustic neuritis. It was this case chiefly that enabled Witmaack to deduce the important conclusion that deafness and disturbances in the labyrinth, occurring in certain diseased conditions and in poisoning by certain drugs, were due, not to circulatory changes (anæmia and hyperæmia) of the labyrinth, but to degeneration of the acoustic nerve fibres "up to the lamina spiralis and the spiral ganglion." It is safe to say that in Witmaack's case the equal and uniform affection of the labyrinth on either side was responsible for the absence of vertigo, although Witmaack found but little involvement of the vestibular trunks. Clinical observations, especially those made with aural irrigation tests, show conclusively that in degenerations of the cochlear nerve the vestibular is also affected, though perhaps to a less extent.

The anatomical and physiological relations of the vestibular nerve to the medullary nuclei and the cerebellum, as well as to the higher centres, have been fully discussed in previous chapters. In seasickness the paths along which impulses originating in the ampullary receptors of the canals, and in the receptors of the otolithic apparatus of the vestibule, are precisely the same as those along which similar impulses travel in rotation- and aural irrigation-sickness, the *modus operandi* being the same in each instance. Enhanced irritability (lowered threshold value) of one set of ampullary or macular receptors, as compared with the condition of the analogous opposing set of receptors in the labyrinth of the opposite side, disturbs the balance of the vestibulo-cerebellar reflex mechanisms, which are constantly in operation, serving to maintain automatically the upright position of the body and to steady the trunk and head upon the fixed lower supports. The result is vertigo, nystagmus, etc., and a disturbance of the normal mechanisms of equilibrium whereby the latter are, to a certain extent, overthrown, necessitating the constant employment of voluntary effort to counteract displacements. In this manner an undue amount of tension is put upon the whole nervous and muscular systems. But besides controlling the skeletal muscles in acts of balancing, the labyrinthine mechanisms stand in close relation to gastric and intestinal movements. Labyrinthine impulses reach the cerebral and medullary centres through the cerebellum, or perhaps in the case of impulses originating in the macular receptors by more direct pathways. In this way labyrinthine impulses affect the vagus, glosso-pharyngeal, and facial motor neurones through the vomiting centre, as well as the vaso-constrictor and other centres of higher and lower levels. In ordinary mild cases of seasickness the primary effect is one of stimulation. Repetition or increased intensity of the stimulus sooner or later causes the well-known functional disturbances in the related organs and mechanisms. The nature and mode of production of these disturbances have been fully discussed in the chapters devoted to rotation-sickness. The inevitable result is a general state of exhaustion, with demoralized circulatory and gastric function and a general weakness and irritability whereby the slightest mental or physical effort causes discomfort and ordinary sights, sounds, odours, etc., become distressing and intolerable.

The exhausting effect of interference with the cerebellum has long ago been demonstrated by Weir-Mitchell,[62] Luciani,[61] and others. Failure and perversion of gastric function become in seasickness a secondary source of distress by causing a deficient supply of nourishment to the body as a whole, and by a constant flow of afferent impulses from the interior of the organ through the vagus to the sensory portion of the dorsal nucleus, where the sensory vagus centre is in close relation with the sensory centres of the facial and glosso-pharyngeal nerves, and with the cerebellum and higher centres. Such afferent impulses originating in the gastric receptors tend to maintain a constant state of nausea and increased salivation, and keep the victim in a condition of extreme wretchedness on the verge of vomiting. Disturbed circulatory function also becomes an important factor, causing in the medullary and cerebral centres a blood-supply that is irregular or insufficient to meet the needs of normal metabolism and the removal of waste products. The use of the eyes, especially in looking at moving objects, is another source of distress; as, under ordinary circumstances, in looking at moving objects, the ocular movements are mainly executed by the oculo-cerebellar mechanisms, aided by the vestibulo-cerebellar mechanisms, both sets of mechanisms using the same final common paths. In seasickness the cerebellar centres, and indeed the whole individual, are in a state of disorganization, with marked inability and disinclination for work of any kind. The well-known influence of odours upon those suffering from seasickness, as well as the perversion of the sense of smell, are to be accounted for by the instability and abnormal irritability of the cerebral, cerebellar, and medullary centres, and chiefly, perhaps, of the vomiting centre and those centres located in the dorsal nucleus. The fibre paths, along which such olfactory impulses may travel, have been already discussed.

Many authors of the present day attribute seasickness in a vague and general way to unaccustomed irritations of the optic, labyrinthine, and kinæsthetic mechanisms. As previously pointed out, under certain conditions, the eyes might possibly be the prime cause of seasickness; but, in the light of our present knowledge, the average eye is not nearly so susceptible to the effects of irregular motions, with sudden stops and starts as the labyrinthine apparatus. In rotations, aural irrigations, and in galvanism applied to the mastoid areas, the individual may be sickened when the eyes are excluded by closing or covering them; whilst if the labyrinth be absent on both sides, or incapable of functioning, neither animals nor men are susceptible. In order to prove that the eyes or any other organ may be the prime cause of seasickness, it would be necessary to exclude first the effects that are constantly present in every case in which there exists even part of a functionating labyrinth. It has been demonstrated in rotations and aural irrigations that disturbance of the vestibulo-cerebellar balanced mechanisms is always associated with nystagmus. The ocular apparatus is, therefore, to a greater or less extent involved secondarily in the labyrinthine irritations which cause seasickness, and is frequently, if not always, involved to some extent in the causation of the distressing symptom, vertigo. All this, however, merely shows that the eye may be, and often is, an important secondary factor in the etiology of seasickness.

The "kinæsthetic sense" is a term designed to cover afferent impulses from muscles, joints, tendons, etc. These impulses originate in the proprioceptors of the various organs. We have seen that the cerebellum by means of its reflex mechanisms adjusts and controls the muscles involved in acts of equilibration, and that it does so by means of its afferent and efferent paths. The chief afferent cerebellar paths are (1) the vestibular, (2) the ocular (including optic), (3) the kinæsthetic, and (4) the cerebral. The cerebellar mechanisms are daily subjected to all sorts of jars and motions by people on shore, and seldom produce phenomena in any way resembling those of seasickness. As before stated, it has been shown by Bickel that, in the dog after section of the posterior nerve roots, the semicircular canals can compensate the loss of spinal afferent cerebellar impulses. As the cerebellum ordinarily controls in a reflex manner many of the acts involved in equilibration, impairment of cerebellar function throws an unusual and exhausting strain on the centres in the Rolandic regions. In seasickness when cerebellar exhaustion has set in, the kinæsthetic mechanisms may be a secondary source of irritation on account of functional disturbance, and may thereby aid in maintaining or aggravating the disorder. In certain movements of the boat, e.g., sudden prolonged upward or downward motions the function of the kinæsthetico-thalamic paths may be involved leading to a temporary loss of coordination in the under supports of the body with the result that the cerebellum has no secure base toward which to make its equilibratory adjustments. It is possible that this may be occasionally a primary factor in the causation of seasickness, but if it ever is it never attains to more than subsidiary importance. In locomotor ataxia, patients standing erect with the feet close together, do not become dizzy or nauseated when the eyes are closed, but simply sway or perhaps fall. This shows the relation the eyes, under certain circumstances, may bear to the optic thalamus and to the coordination of the feet and other members. Here again is another way in which the eyes in seasickness may be unusually strained, and ultimately become a secondary source of irritation and exhaustion. Helmholtz[283] says: "One feels the traction of gravity (on board ship) now apparently to the right, now apparently to the left, now forwards and now backwards, because one is no longer able to find with his eyes the direction of the vertical. Only after long practice, as I myself can testify, does one come to use gravity as an exclusive means of orientation, and only then does the vertigo cease." Regarded from any standpoint neither the eyes nor the kinæsthetic sense can be seriously considered as more than secondary, or at most subsidiary primary factors in the causation of seasickness. Almost any organ or function may be so affected as to become prominent as a symptomatic feature and source of distress. It is to misinterpretation of such occurrences we are indebted for the many theories that attribute seasickness to any one of the numerous secondary factors involved. Throughout the ages, seasickness has been much of a puzzle to mankind. The first step toward the actual solution of the problem was made by Flourens in 1824, when he divided the semicircular canals in pigeons and rabbits.

CHAPTER XXXIII

ON THE OCCURRENCE OF SEASICKNESS

The literature of seasickness teems with conflicting reports, as to the immunity or susceptibility of certain groups of individuals, the cause being failure to recognize the various shades and degrees of severity of the condition. It is commonly agreed that those of the nervous or neurasthenic type are more susceptible than those of coarser fibre (Beard[242]) but little reliance is to be placed on such distinctions since, as we have seen, very neurotic individuals with degenerated labyrinthine nerves may show absolute immunity. Children, most authors agree, are comparatively immune. Here again is room for doubt. Children old enough to run about, are frequently affected, but as a rule recover rather quickly owing perhaps to the mental and physical conditions peculiar to childhood. Very young children, such as infants in arms, are seldom sick, either because they are not called upon to perform much in the way of acts of equilibration, or because of the undeveloped condition of the mechanisms of equilibration. It must be remembered, however, that children cutting teeth suffer occasionally from vertigo and nystagmus and that infants have been actually seen to suffer from what was undoubtedly seasickness. In children it frequently happens that an extreme condition of nervousness develops at sea. Thus a girl of eight suffered greatly from inability to rest or relax her feet properly in bed at night, and constantly cried out to her mother that she did "not know what to do with her feet for they wouldn't keep still in any position." This symptom will be readily appreciated by those in whom overtense conditions of the nervous system do not, at times, permit proper relaxation of the muscles.

Old age is frequently immune for numerous reasons, but more particularly because of the rule that in advancing years there is a tendency to arteriosclerosis and to bilateral atrophy and degeneration of the auditory nerve. Those suffering from chronic wasting diseases and from continued excesses in alcohol also seem to have a relative immunity. Here the cause is deterioration of the vestibulo-cerebellar mechanisms whereby they do not so readily respond to labyrinthine stimulation and depression, or deterioration of the cerebral and medullary centres whereby the labyrinthine irritation is less effective in evoking the phenomena of the malady in their full severity. And here it is significant that in any one of these conditions degeneration of the auditory nerves may occur as Witmaack[26] has shown.

Some apparently normal individuals appear to enjoy immunity, the cause being peculiarities of structure or function of the vestibulo-cerebellar mechanisms or of the related cerebral and medullary centres. Similar immunity to a certain degree has been observed in some individuals in experiments with rotations, aural irrigations and galvanism. Such individuals have a natural or acquired power of resisting, to a certain degree, the effects of certain forms of stimulation peculiar to seasickness just as ordinary individuals offer differing degrees of resistance to various other forms of stimulation by alcohol, tobacco, pain, etc. Probably no one with functioning labyrinths is totally immune to seasickness under all conditions of circumstance and weather. It is certain that no one is immune to rotations of all grades of severity unless both auditory nerves have degenerated, or both labyrinths have been excised as in many of the experiments which have been performed upon animals. It is a common popular error to suppose, that because an individual is robust and healthy he will not therefore fall a victim. Seasickness in ordinary conditions of health is primarily the response of the organism to repeated and irregular forms of labyrinthine stimulation. Predisposition to seasickness, therefore, merely implies ready, alert reflex mechanisms, and is in no way directly related to the health or strength of the individual in so far as its primary causation is concerned. In fact so common is seasickness amongst normal individuals that the burden of proof rests with those who are immune to prove that they are free from abnormality.

Trainers of athletes of extensive experience and of world-wide reputation have given the information that seasickness occurs amongst sprinters, distance runners, weight throwers, jumpers and football players alike indiscriminately. Several athletes professional and amateur were consulted, and the information obtained corroborated that of the trainers. The sea has frequently taken the "edge" off athletes in condition, so that after the voyage it took some time, in some instances a long time, before the individuals came up to their usual performances. One individual, a professional pugilist whose chief merit consisted in his extraordinary ability to "withstand punishment" without being "knocked out" sent in a facetious reply in which he expressed the opinion that "wet goods" (alcohol) had more to do with the causation of seasickness than any other factor. This individual was very probably immune to seasickness and his statement regarding the relation of alcohol to the causation of seasickness, though flippant in form, in all probability represents conclusions drawn from personal experience. Another pugilist noted also for his ability to resist "the knock-out" blow reported that he had never been affected by seasickness. This individual, however, has descended from a long line of ancestors famed for their exploits on sea.

Several professional equilibrists, acrobats and tumblers were interrogated by mail. Not one reply was received. This fact inclines one to the belief that men of these callings are prone to seasickness and consider, perhaps, that it might be a reflection upon their professional ability to own up to the fact. A famous distance runner who holds many records also failed to send in a reply. The author saw this man in the throes on a transatlantic trip. It is well known, however, that famous athletes of every description are subject to seasickness.

The question as to the immunity of animals has been dealt with extensively in the literature to which it lends an amusing and, at all times, interesting feature. Here, as elsewhere, a great difference of opinion prevails. The general consensus is that most, if not all, animals suffer from the malady. The opportunity of studying the question directly, not having presented itself, inquiry was made from those who not only had the opportunity, but were otherwise qualified to form an opinion. Dr. G. G. Flemyng of the White Star Liner *Teutonic* was surgeon in charge of the vessel which brought over to the London Zoölogical Gardens the Australian collection of animals amounting to about 5,000 specimens. The doctor stated that most of the animals came down with common ordinary seasickness just like human beings and many died from the malady.

A fellow traveller whose business was concerned in taking horses from America to Europe and selling them there, was next interrogated. This individual stated offhand that animals do not get seasick. Upon closer questioning he stated that his experience was confined to horses of the draft variety and he qualified his former statement by saying that animals do not get seasick—if you know how to feed them properly. It was later discovered that this man's conception of seasickness was a disturbance of function of such intensity as to threaten the life of his animals.

CHAPTER XXXIV

THE EFFECTS OF SEASICKNESS AND THEIR RELATION TO DISEASED CONDITIONS

The effects of seasickness upon the nervous system and upon the circulatory and digestive functions have been fully set forth and commented upon in those chapters dealing with observations on seasickness.

A glance at the protocols will convince the most skeptical that seasickness occurring in normal individuals throws an enormous strain upon the circulatory mechanisms and especially upon the heart which more and more has to assume the burden of maintaining the blood-pressure as the vaso-motor system becomes exhausted. And not only is the heart overworked but on account of failure of the digestive functions it is usually under-nourished and frequently even starved. Look at the question as we may, whether we attribute the condition of the circulation to stuffy cabins, warm weather, the motions of the boat or to whatever cause, the effects upon the heart are always a matter of grave concern. And here the question arises: If these things occur in healthy adults, how are the effects of seasickness upon the degenerated heart muscle or upon a generally enfeebled organism to be estimated? No one would think of allowing an individual of middle age and with a diseased or enfeebled heart muscle to engage in a physical contest where the strain might be severe and prolonged. And yet it is a common thing for those recovering from protracted or severe illness to be sent to sea to complete the restoration to health, without the practitioner giving serious thought to the special strain that a voyage might entail upon the organism and especially upon the heart. Many individuals past the meridian of life, have been seriously injured by injudicious travelling on sea. It is scarcely an exaggeration to say that a large percentage of the deaths that occur at sea are attributable directly or indirectly to the effects of seasickness. Sometimes it is said that seasickness is not an unmitigated evil and that in certain cases it may do an individual good. Such a statement may perhaps be occasionally permissible for its psycho-therapeutic effect, but it would be a difficult task to convince any unbiassed mind that the conditions revealed by the protocols representing actual observations taken at sea, could, in any way whatever, be a benefit to the individual. On the contrary, it is manifestly apparent that they might permanently injure him and in presence of actual disease, whether acute or chronic, so handicap the patient as to seriously mar the prospects of ultimate complete recovery.

The effects of seasickness upon pregnant women are frequently alluded to in the literature, many cases of abortion, miscarriage, and premature labour being cited. There is a group of cases of amenorrhœa which gynecologists attribute to change of climate. The condition develops usually in domestics of foreign birth and makes its first appearance when the individual leaves her home to earn her living in distant lands. Judging from the frequency of amenorrhœa after sea voyages and remembering the exposure to which steerage passengers when seasick are submitted as well as the profound impression which seasickness makes upon the nervous system and indeed upon the whole organism, it is perhaps more reasonable to attribute this form of amenorrhœa to the effects of seasickness and exposure rather than to mere change of climate.

The effects of seasickness on the nervous system have been frequently alluded to in previous chapters. The psychic depression is frequently so extreme, and cerebral function so completely perverted, that self-control becomes an impossibility. Many of the numerous cases of suicide that occur at sea, have for their immediate cause this psychic depression. Suicide on land, like other grave errors in conduct, is seldom the result of sudden inspiration. The thought of it has previously been entertained and examined. Comparisons have been instituted and some sort of hypothetical conclusion reached as to its feasibility under certain circumstances. Every individual at some time or other, turns over in his mind the thought of suicide and the mental attitude he assumes toward it, though it appears trivial perhaps at the time, may, later on, become a matter of the gravest import in conditions of psychic derangement. Many go to sea, with no thought of suicide beyond these hypothetical conclusions, retained as subconscious memories, and in the psychic depression induced by a mild grade of seasickness, the incapacitated mind erroneously concludes that the requisite conditions have arisen which, according to previous reasonings, render suicide feasible and proper. Thus it happens that individuals pay with their lives the penalty of poorly regulated unsystematic habits of thought. Thoughts in a great measure make us what we are and it is the individual's business in every instance to see that his thoughts do not tend to unmake himself or others.

From these considerations it is manifest that a serious responsibility rests with the physician who recommends sea voyages in conditions in which the "reserve margin" of any of the organs is encroached upon by disease or faulty habits of living. In disordered states of the nervous system, especially where, in the history of the individual, there is the least intimation of manic-depressive or other form of insanity, extreme care should be taken in recommending sea journeys, if disastrous consequences are to be averted.

The number of suicides committed at sea, and the carelessness with which unfortunates suffering from seasickness are treated is a disgrace to civilized humanity. The lonely individual suffering inner torments, as the result of repeated labyrinthine irritations, wanders about in a state of mental agony, not knowing what is the matter with him, nor how to properly orient himself in the whirl of his disordered imaginings. Instead of receiving proper medical treatment which would include close supervision, and a goodly amount of reassurance, such an individual is left severely alone, with the result that relief from mental distress of purely artificial origin is sought in self-destruction.

Although much has been done in recent years to improve the general conditions of travelling at sea, yet very little has been done to alleviate the distress of those suffering from seasickness. The blame for this does not perhaps wholly lie at the door of the ship-owners, since so little has been hitherto definitely understood about the malady. However, the fact remains that the hospital on ship board is generally located in the stern, over the propeller, where the motions of the boat have the most sickening effect and where the jolting of the machinery is a constant cause of continued distress. The sick and suffering are committed to such quarters when their condition has become extreme. Moreover, one physician can hardly be expected to properly look after the needs of two or three thousand persons. In addition ship surgeons should receive special training so as to thoroughly understand the effects of seasickness upon the physiological economy, that they may be enabled to treat intelligently not only seasickness, but all other illnesses that may arise during a voyage. Medical colleges also should give instruction on this subject which is, at least, as important to the general medical man as "insurance examination" and other subjects at present receiving special attention.

It seems strange that in this age of magnificent philanthropy, no one has seen fit to inquire into this matter, with a view to improving the hospital facilities on ocean liners and the quantitative and qualitative efficiency of the medical attendants. At the present time, perhaps, no other field offers such great opportunities for the relief of human suffering and the promotion, on a large scale, of the comfort of the many both rich and poor alike.

The possibility of seasickness resulting in subacute or chronic impairment of the digestive functions and even in chronic inflammatory conditions of the gastric mucosa has been referred to in a previous chapter. The extreme degree of protracted congestion of the abdominal viscera undoubtedly may cause a predisposition to varying grades of inflammation not only in the stomach, but also in other organs. It is very probable that prolonged congestion of the ovaries is an important factor in causing the amenorrhœa that follows seasickness.

The effect of seasickness upon the nervous system often manifests itself in disturbances in the growth and nutrition of the hair which often tends to fall out during a voyage and frequently remains sickly and "unmanageable" for some time after the journey has ended. The general effect upon the nervous system is also reflected in the obstinate constipation which, however, involves other factors such as deprivation of food, failure of gastro-intestinal motility, etc. During the height of an attack of seasickness the urine is also diminished and concentrated, due in part, to deprivation of liquids, failure of absorption or both, and in part to disturbance of the nervous and circulatory mechanisms.

It has been frequently stated that "colds" are an unusual thing at sea. Barker[239] states that he has known them to have occurred. It is a fact, however, that they are unusual. Subject "B" of these studies has never "caught cold" at sea, although it has been his custom to sit on deck without extra clothing in all sorts of weather. Indeed it has been his experience that if he "had a cold" going on board, it rapidly disappeared in a short time after putting out to sea.

One thing should always be remembered by those whose means do not permit them to travel in a separate stateroom, and that is, that contact is usually very close between the occupants of the same cabin, so that if one occupant is affected with a communicable disease, the others may possibly become infected. At the present time quarantine supervision is so strict, and the laity know so much about disease, that infection occurring in this way must be exceedingly rare, and yet first-hand evidence convinces us that there is such a possibility. As late as the autumn of 1906 a gentleman in a hurry to get back to the United States found it difficult to procure accommodation in the first class. He telegraphed to various lines, and finally was pleased to learn that a certain line had a vacancy which he immediately secured. On the first night out at sea, he found the cabin small and occupied by four full-grown men. One of the occupants coughed all night so that sleep was impossible except in snatches. This annoyance was borne in a good-natured way by the occupants of the cabin, but early daylight revealed the uncomfortable fact that the individual with the cough was an undoubted victim of pulmonary tuberculosis, and what was still worse, that he acted as though he was utterly unaware that he was a menace to his fellow room-mates. Application was quietly made to the purser but without redress. The captain was then approached, and to his credit be it said that he did everything in his power to relieve the situation. The hospital was placed at our American friend's disposal, but as he was very prone to seasickness he feared the excessive motion and the vibration from the machinery, for the hospital was located just above the propeller. The printers' room was next suggested. Investigation showed a state of affairs that precluded it from acceptance. Finally with the aid of the ship's carpenter a bed was arranged over a bath-tub in a clean, wholesome bathroom. During the remainder of the voyage, the sick man coughed and coughed, and apparently never made the slightest attempt to intercept his column of spray at times painfully visible in the light that streamed from the port-hole. His two roommates had to make the most of the situation, although one of them was fully aware of the danger, for he himself had been a victim of tuberculosis, and was then actually returning from a protracted sojourn on the Italian Riviera, whither he had gone to rid himself of the disease. Another important fact is that this tuberculosis victim, the one who coughed constantly, was a steady reader. Book after book came to him from the ship's library to be thumbed and coughed upon from cover to cover. This should supply food for thought for travellers accustomed to delving into the volumes of the ship's library. It may be true that colds are unusual at sea, but close contact for whole nights in small stuffy cabins with closed port-holes, backed by the depression and exhaustion that accompany seasickness, render infection an easy matter under such conditions as those just related.

CHAPTER XXXV

HOW RECOVERY FROM SEASICKNESS TAKES PLACE. OUTLINES OF TREATMENT

The manner in which recovery from seasickness takes place, merely illustrates the wonderful adaptability of the organism to circumstances and conditions. The cerebral cerebellar and medullary centres become accustomed to the degree and rhythmicity of the labyrinthine stimuli to such an extent that the cerebral centres are no longer preoccupied and perturbed by the unaccustomed irregular activities of the cerebellar functions of equilibration and cease to hamper the latter by attempts at compensation, thereby permitting the cerebellar mechanisms to readjust themselves to the new conditions. The medullary centres, having somewhat recovered from the surprise of unaccustomed stimuli, proceed with the business of carrying out their proper functions regardless of afferent labyrinthine impressions of the particular degree and rhythmicity experienced. Recovery does not take place by the labyrinthine receptors becoming "accustomed to the motions of the endolymph" as some authors assert. It is in the nervous centres that the readjustments take place which are responsible for the recovery, just as it is in the centres and not at the periphery linger those impressions which are responsible for the existence of sea-legs upon shore for many days after a voyage has ended.

In seasickness as in other ailments, the great desideratum of the profession and laity is the prophylaxis. From the experiments and observations set forth in these pages and from an extensive experience with the malady at close range, it is safe to assert that seasickness can, in most instances, be effectively prevented by the adoption of judicious measures. supported in certain cases by the use of certain drugs administered with proper precaution as to time, dosage, age of the patient, etc. Healthy individuals might perhaps be permitted to procure and use such drugs, but the practice is not in all cases to be recommended, the chief reason being that in certain diseased conditions of the organs and in certain phases of seasickness drugs and circulatory stimulants of any kind may be contraindicated.

Theoretically, the first indication in the prophylaxis is, of course, to lower the irritability of the labyrinthine nerve terminals or of the vestibulo-cerebellar mechanisms. It must be confessed that in the trials made thus far, no safe drug has been found to meet this indication. Moreover physiological considerations lead to the belief that such a drug, if found, might be of doubtful benefit as a pure preventative, however it might otherwise help in the actual treatment of seasickness. Failing to prevent those irritations of the labyrinthine receptors the next indication is to prevent their pernicious effects upon the cerebellar, medullary, and cerebral centres. Here many measures are available including general hygienic measures preparatory to the voyage, mental quiet, attention to the gastrointestinal functions and the employment of drugs, especially atropin or hyoscyamin preferably by hypodermic administration. These drugs, however, should not be used indiscriminately nor continuously. Bromides have little to recommend them. The use of cocain is contraindicated. Clinical and experimental results obtained in seasickness and in rotation sickness, and the peculiar effects produced by the drug upon animals in which it causes *manège* (circus) movements, lead to the belief that cocain predisposes the organism to seasickness. In one instance at least the administration of cocain by a dentist has led to all the phenomena peculiar to profound labyrinthine disturbance (Koenig[228]). The size, location, and ventilation of the cabin or suite has an important bearing in prophylaxis. The avoidance of hurry previous to going on board and congenial adaptation to the new environment, are a distinct help in prevention, whilst rest in recumbency has been justly advocated in all times. Light and easily digested food, taken in moderation, and under conditions most favourable to digestion, is of benefit. Hypnotic suggestion as a preventative and even as a means of treatment of a present attack has been tried with varying success. Beard[99] believed that it could prevent rotation sickness. Experience with hypnotism in the experiments in rotation, and aural irrigations shows conclusively the limited benefit that is to be expected from suggestion in these conditions and in true (somatic) seasickness. Where the psychic factor predominates, or seems to be the chief source of distress, hypnotism is of undoubted benefit; but where the condition is one of true (somatic) seasickness caused by labyrinthine irritation little can be expected from hypnotic suggestion beyond the alleviation, to a certain extent, of disturbances arising from some of the secondary sources of irritation. In this respect, therefore, hypnotism has a value in true seasickness that puts it on a par with the thousand and one remedies that have been put forward by writers from time immemorial as sure and certain cures. It is, however, very questionable whether the influence of post-hypnotic suggestion would continue sufficiently long even in cases of so-called psychic naupathia to be of lasting benefit. Hence it would be necessary to have the individual hypnotized again and again at frequent intervals and the suggestions repeated. These and like considerations lead to the conclusion that hypnotism has a limited field of usefulness in seasickness, and that it is perhaps a therapeutic resource which should be reserved to meet certain indications only. This does not mean, however, that ordinary methods of suggestion should not be utilized to brighten and enliven prospective and actual travellers. Suggestion is a potent means of breaking up the vicious train of secondary psychic phenomena and should be made the most of under all conditions and at all times.

Various devices have been employed to prevent seasickness. Swinging beds have been used, but evidently little benefit was obtained, for their use has been abandoned. Since the introduction of the mono-rail system of railway, various suggestions have been made to apply the principle of the gyroscope so as to obviate the rolling motions of ships at sea and thereby to prevent seasickness. It is difficult to see in what way such a thing could be effected so as to restrain all the movements of a ship, but stranger things have come to pass, and one never really knows what can be done until some genuine attempt is made.

The treatment of an actual attack of seasickness resolves itself into combating the effects of primary labyrinthine irritations upon the cerebellar, medullary and cerebral centres and in eliminating, as far as possible, all secondary sources of irritation. In most cases seasickness is protracted and aggravated by bad management.

Atropin, given preferably hypodermically, has been found to counteract the psychic depression that accompanies seasickness. In addition, by its action in depressing the sensory nerve terminals all over the body, and especially in the stomach and intestine, it effectively cuts off afferent irritating impulses which are such prominent secondary sources of distress. By its well-known action upon the medullary centres atropin can be of wonderful service in regulating to a certain extent, some of the circulatory disturbances incidental to seasickness, but the observations on the circulation, as shown in the protocols, indicate that in protracted voyages and where vaso-motor exhaustion has already set in, atropin or any method of stimulation has to be used with caution.

The stomach is the most important of the secondary sources of irritation in seasickness. The care of this organ in seasickness differs in no wise from the care that should be accorded it in conditions of acute or subacute gastritis on shore. It has been shown that an extreme degree of congestion of the gastric mucous membrane is present in seasickness, hence the stomach-tube should not be used at all, or used only with extreme caution. The liberal blood-supply of the gastric submucosa and the existence of the œsophageal plexus of veins should be remembered. Slight trauma in these regions has been followed by fatal hemorrhage, and fatal gastric hemorrhage has been known to occur in seasickness and other conditions in which violent vomiting occurs. Hence if the stomach needs to be washed out it is best to have the patient drink warm water with some harmless alkali (bicarbonate of soda) dissolved in it. This solution helps to separate the masses of mucus from the gastric walls, and it is readily vomited. During the acute stages of gastric irritation food of every kind is contraindicated for a short period. Later on liquid nourishment may be given tentatively. It is important to get the patient to take food as early as practicable, and of the different foodstuffs the first preference should be given to some digestible form of the carbohydrates. The absence or reduction of free HCl noted in the analyses of the various test meals, renders it inadvisable to give meat in any quantity, until the stomach functions are fairly reestablished.

In certain conditions extracts of the gastric mucosa or meat-extracts such as beef-tea, etc., will find indication. These extracts may be given *per os* or *per rectum*. It is not advisable to administer them intravenously or by hypodermoclysis, as in the author's experimental trials the subjects were invariably affected with fever and symptoms similar to those of serum sickness. Undoubtedly in many instances the extracts may do good, especially in conjunction with atropin and strychnin in grave cases, both on sea and land, where the restoration of gastric and intestinal function is urgently demanded. It is to be added, however, that in some of the tests in which atropin had not been previously administered the extracts seemed to add to the irritability of the gastric mucosa aggravating the subject's distress.

Constipation may be obviated by a judicious use of cathartics in ordinary cases. The hormones (peristaltic hormone of Zuelzer) have been used on shore for the relief of constipation in various conditions (Zuelzer,[286] Saar,[287] Unger,[288] and others) and much is to be hoped from their use in seasickness, especially when combined with the other measures recommended, viz., atropin, etc. It is earnestly to be hoped that some preparation of secreting hormone adapted to hypodermic or intramuscular administration, will be forthcoming soon, as the author's experiments have led him to believe that the hormones will prove a decided help in the treatment of severe cases of seasickness.

Disordered circulation is another great secondary source of irritation in seasickness. In order to meet properly the indications under this head, frequent observations of the pulse and blood-pressure should be made. The physician should thoroughly understand the peculiarities of the individual's circulatory mechanisms. In any individual over forty, and in persons who have been a *bon vivants* all their lives, the greatest care has to be taken to avoid injury to the cardiac musculature. Such individuals should not, under any circumstances, undertake a journey upon the seas, without consulting their physicians on shore, who should carefully instruct them regarding matters calculated to affect the present or future efficiency of the heart. Rest in bed may be the indication when the circulatory function has been carefully studied. The patient may chafe at this, but in common honesty he should be instructed as to the consequences. Few individuals would be willing to barter years of health and efficiency for the gratification of going upon deck to show what good sailors they are. And yet that this must be a matter of frequent occurrence may be readily deduced from a careful study of the figures given in the protocols. As before stated, the conservation of the individual's health in regard to this matter rests in a great measure upon the instructions given by the medical adviser on shore. As the circulation improves measures calculated to benefit the general condition may be employed with care. Cold bathing should always be prohibited until a study of the circulation shows that it is permissible. The same has to be said of exercise and of any remedy that tends to stimulate the cardio-vascular apparatus. So long as there is evidence of exhaustion in the neuro-vascular mechanisms, circulatory and vasomotor stimulants are contraindicated. Fresh air is always of importance, though this does not necessarily imply that the patient is to have a cold air bath. The protocols show what a depressing effect upon the neurovascular apparatus a hot cabin may have, and where the object is to prevent strain and injury to the heart, the temperature of the cabin should be regulated as far as possible, and by artificial means if necessary.

The other sources of secondary irritation in seasickness are the eyes, the ears, the olfactory organs, the psychic cerebral areas, the spinal cord, and the general peripheral sensory terminations. In the condition to which seasickness may reduce any individual, the active functioning of any of these organs may be a source of depression and distress. Thus the prolonged use of the eyes is to be forbidden as in reading steadily. Noises also should be suppressed. Offensive odours are to be avoided, and this makes one wonder why gentlemen will smoke on deck where the odour they create sends shivers down the spines of delicate, suffering women. Anything that tends to unduly arouse the emotions, or to evoke a prolonged train of serious thought should be avoided. The brain should be rested as much as possible, and the individual placed in the most agreeable and inexacting surroundings. It frequently happens that in certain stages of seasickness the sexual desire is exalted out of proportion to the actual power of the individual. Under such circumstances attempts at intercourse should be prohibited.

From the results obtained in rotation sickness, the conclusion was reached that hyoscin should never be used in seasickness. Morphin was also found to have disagreeable effects notwithstanding its effect in enhancing the circulatory function. Any drug or remedy that tends to depress the medullary centres should be avoided in seasickness and those drugs and measures which tend to stimulate these centres must be used with caution and with due respect to the condition of the centres at the time, i.e., whether they are in a state of exhaustion from overirritation or merely in a state of lethargic inactivity.

The gyroscope has been mentioned as a possible means of preventing seasickness by restraining the movements of the ship as a whole. In the actual treatment of seasickness or of any serious illness at sea, the principle of the gyroscope might be applied in conjunction with a swinging deck or compartment. Thus, for instance, a swinging hospital steadied by revolving fly wheels, might be maintained on every large steamer for those who may be dangerously ill. Such a contrivance if practicable, would also render surgical procedures at sea much safer than they are at present and would aid convalescence materially besides reducing the patients' suffering to a minimum. Recently, a system of U tubes or tanks devised by Frahm has been tried on some of the ships in the German navy. The reports indicate that the rolling of the vessels was considerably reduced. Whether the use of such tubes will prove effective in preventing seasickness is a matter for further research. Such devices need not necessarily completely stop all rolling or movement of the boat, as it will materially aid sufferers at sea if the sudden stops and starts in the ship's movements be eliminated or even partially restrained.

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INDEX TO THE SEMICIRCULAR CANALS

Abdominal and pelvic viscera, fibres for blood-vessels of, 100
Abdominal viscera, congestion of, 177
  severance of, from spinal cord, 103
Abducens centre, 20
Abducens nucleus, fibres from, 49
Abducent nucleus, fibres to, 40
Absorption, controlled by local mechanisms, 174
  experiments, 173
  of food, active, 234
  tests for, 176, 189
Accelerator centre, location of, 102
Accelerator fibres for the heart, 102
Accessory acoustic nucleus, 14
Accessory nerve (the eleventh), a motor nerve, 35
Accessory olivary nuclei, connections of, 56
Accommodation constriction, 110
Accommodation synkinesis, 110
Acoustic area, elements of, 9
Acoustic nucleus, constitution of, 14
Acoustic striæ, component fibres of the, 16
  fibres from the, 53
Afferent cochlear fibres, 20
Afferent and efferent impulses, relay station for, 41
Afferent paths, 45, 46, 51, 52, 53 54, 56, 59, 60, 62, 67, 68, 72, 73
  peripheral terminations of the, 83-94
After-nystagmus, apparent movements of objects during, 262, 267, 274
  horizontal, 276, 277, 279
  premature induction of the natural, 195, 204, 205
Ampulla, of the superior canal, irritation of, 5
  of the posterior canal, 5, 6
Ampullary receptors, muscular associations of, 281, 282
Anæsthesia, recovery from, 317
Anatomical connections, synopsis of, 40-77
Anelectrotonus, state of, 280
Angular gyrus, left, 78
Animal experimentation, 265
Animals live after severance of abdominal viscera from spinal cord, 103
Anode tests, 270, 271
Ansa lenticularis, position of, 64
Ansa peduncularis, position of, 64
Anterior nerve-roots, connections of, 100
Apertura externa aquæductus, position of, 4
Aqueduct of Sylvius, grey matter of, 69
Aquæductus vestibuli, 7, 37

Arcuate fibres, anterior and posterior superficial, 43
Arcuate nucleus, fibres of, 44
Arterial relaxation, shock due to, 134
Arterial tension, nature's methods in combating lowered, 135
Arterio-sclerosis, general, 246
Association fibres, division of, 74
Atropin, effect of, in relieving stomach discomfort, 189
  action of, on the cerebral psychic and motor areas, 189, 190
  influence of, in rotations, 134
Auditory area, stimulation of, in the temporal lobe, 15
Auditory canal, cold irrigations of the external, 245
  effects of irrigations of the external, 207
  internal, relations of nerves in, 12
  sensory stimulation of the external, 248
Auditory nerve, elective vulnerability of cochlear branch of, 38
Auerbach's plexus, cells and fibres of, 165
Auer's experiments on the splanchnics, 169
Aural irrigations, cause of nystagmus in, 243
  disturbances of coordination accompanying, 248
  effects of, 181
  effects of, upon the eyes, 250
  effects of, upon the digestive apparatus, 233-249
  effects of, upon equilibration, 237
  rise of blood-pressure in, 243
  general effects of, 207-219
  mechanism of, 288
  phenomena of, 219, 247
Aural irrigations, possible aid in the treatment of nerve deafness, 249
  pylorus tightly closed in, 235
Autophony, 246

Bahnung, mutual reinforcement of impulses, 177, 179, 180
Bannister's researches and experiments on the semicircular canals, 125
Bilateral cortical innervation, 31
Bilateral hot irrigations, 244
Bilateral irrigation, 275, 279
Bipolar neurones, 13
Blood-pressure, changes in, during irrigations, 255
  effect of strychnin and atropin in maintaining the, 134
  lowering of the, 102
  mechanism which regulates the, 133
  raised by vaso-constriction, 133
  rise of, in aural irrigations, 243
  threatened dissolution from fall of, 134
Blood-supply of the labyrinth and of the cranial nuclei, 37-39
Blood-vessels, contraction and dilation of, 131
  disease of, 246
  fibres for, 100
Blood, vomiting of, in aural irrigations, 233
Bogen-commissure of Stilling, 70
Bowels, uneasiness of the, during rotations, 181
Brain, medullary substance of, 74
  sensory paths of, 72
  vascular supply of base of, 39
Breuer's suction, 267

Bronchial musculature, motor fibres for, 101
Bulbar autonomics, 31

Cajal, tract from the interstitial nucleus of, 86, 87,
Calcarine fissure, 78
Canaliculi cochleæ, venous blood-draining into, 37
Canalis reuniens, location of, 7
Carbohydrates, acid hindrance to digestion of, 175
Cardiac acceleration, 102
Cardiac arhythmia, in rotations, 135
Cardiac fibres, inhibitory, origin of, 34
Cardiac mechanism, sensitiveness of the, 135
Catalepsy in the standing position, 216
Cataleptic rigidity, state of, 217
Cathode tests, 270, 272
Cats, Ferrier's experiments on, 298
  Parson's experiments on, 112
  stimulation of cerebellum of, 291
Caudate nuclei, fibres of, 63
Caudate nucleus, fibres from, 67
Cells of chief nucleus, dislocated groups of, 23
Central nervous system, division of, 90
  continuity of, 98
  efferent paths from the, 99
Cerebellar areas, Ferrier's experiments on stimulating, 302
Cerebellar connections, 43–44
Cerebellar cortex, fibres from, 54
Cerebellar hemisphere, relations of, 114
Cerebellar mechanisms, activity of, 116, 302
  efferent limbs of, 303
  and migraine, 336
Cerebellar mechanisms and neurasthenias of various forms, 336
Cerebellar paths, recapitulation of the, 94–97
Cerebellar superior olivary tract, an efferent cerebellar path for ocular movements, 26
Cerebellar tonus, depression of, 261
Cerebello-cortical circuit, disturbance of, 311
Cerebello-superior olivary tract, fibres of, 57
Cerebello-tegmental tract, fibres of, 47
Cerebellum, connections of the, through rubro-spinal tract, 47
  connections of the, through the middle peduncle, 45–49
  diagrams showing the effects of various operations upon, 324, 325
  electrical stimulation of, 289
  extirpation of lateral lobe of, 323
  Ferrier's stimulation experiments on the, 115
  fibres from the, 56
  fibres passing to the, 34
  paths of the, 26
  pupillary centres in, 260
  relation of the, to specialized sensory organs of equilibration, 115
  stimulation of various parts of the, 290, 291
  transmitting afferent impulses to the, 19
  unilateral lesions of the, 326
Cerebral apoplexy, turning of eyes in, 116
Cerebral centres, direct tonus influence of, 115, 116
Cerebral cortex, auditory paths of, 16
  fibres from, 53

Cerebral cortex, ocular movements in, 294
removal of areas of, 323
Cerebral hemorrhage, confined to one hemisphere, 116
conjugate deviation in, 312
Cerebral motor cortex, 31
Cerebral ocular area, effect of removal of, 322
Cerebral peduncle, arteries supplying, 39
Cerebral synkinesis, phenomenon of, 111
Cervical sympathetic, stimulation of, 257
Chorda tympani, 30
taste fibres of the, 30
Circulatory depression, 134
vomiting in severe states of, 167
Ciliary ganglion, nature of cells in, 107
removal of, 107
Clarke's column, vesicular cells of, 34, 35, 81
Cochlear artery, 37
Cochlear fibres, description of, 13, 14
Cochlear ganglion, ganglion cells of the, 38
Cochlear nerve, disease of, 246
Commissura hypothalamica, fibres of, 49, 68
Commissura posterior cerebri, 66
Commissural tracts, 58
Commissure of Meynert, fibres through, 50
Conceptualization and hearing, 78
Conjunctiva of both eyes, congestion of, 250
Connections of the cerebellum, 43–44
of the cerebellum, through the middle peduncle, 45
of the cerebellum, through the superior peduncle, 46
of the corpus striatum, 73
of the corpus subthalamicum, 67
Connections of the inferior colliculi, 53
of the inferior olivary nucleus, 54
of the lateral fillet or lemniscus lateralis, 52
of the lateral geniculate body, 52
of the mammillary nucleus, 68
of the median geniculate bodies, 54
of the mesial fillet or lemniscus, 49
of the optic thalamus, 62
of the pontine nucleus, 60
of the red nucleus, 60
of the rhinencephalon, 72
of the substantia nigra, 67
of the superior colliculi of the corpora quadrigemina, 51
of the superior olivary nucleus, 56
of the vestibular nerve, 40–43
of the worm (vermis), 58
Cochlear, the nerve of hearing, 12
Cochlear nerve, elective vulnerability of, 246, 247
Cochlear nuclei, fibres from, 56
Cochlear receptors, 246
Cold irrigations, effect of, 233
of the external auditory canal, 245
Column, meaning of term, 79
Commissura arcuata anterior of Hannover, 70
Commissura habenulæ, 65
Constrictor tonus, inhibition of, 259
Control test meals, 235
Coordination, disturbances of, following aural irrigations, 244, 248
Corona radiata, 29, 31
stimulation of, 112, 316
Coronal plane, rotations in the, 194, 203

Corpora quadrigemina, fibres to, 46, 50
  irritation of, 167
Corpus callosum, 69
  fibre paths of, 75
  section of the, 112
Corpus striatum, connections of the, 73–78
  grey matter of, 73
Corpus subthalamicum, connections of the, 67
  grey matter of, 67
Corpus trapezoides, formation of, 14
Cortex cerebri, 28
Corti, organ of, 13
Cortical centres, obstruction of circulation in and vertigo, 39
Cortical paths, decussation of, 27
Cortico-bulbar tracts, 76
Cortico-geniculate tracts, 76
Cortico-pontine tracts, 76
Cortico-rubral tract, fibres of, 76
Cortico-spinal tracts, position of, 76, 77
Cranial motor nuclei, fibres from cerebral cortex to, 29
Cranial nerves, 22
  liable to pressure from exudations, 28
  situation of, 22
Cranial nuclei, blood-supply of the, 37–39
Crista acustica, 8, 10
Crista vestibuli, openings in, 3
Crus cerebri, 31
Crustal fillet, 29, 31, 50
Cuneo-calcarine cortex, lesions of, 78
Cuneus and calcarine fissure, 78

Darkschweitsch, nucleus of, 24
Deafness, senile, cause of, 37
Deiters' nucleus, 42
  fibres from, 55
  neurones of, 19
  tract from, 87
Dentate nucleus, fibres from, 58
Depressor fibres in man, 102
Descending tracts, ascending paths of, 89
Descending vestibular root, termination of, 43
Diagonal plane, effects of rotations in the, 196, 205
Diencephalon, main divisions of, 62
Digestive apparatus, effects of aural irrigations upon the, 233–249
  effects of rotation upon the, 164-182
Digestive functions, gastric, importance of, 167
Digitalon, the effect of, in rotation, 137
Dilator contractile tissues, excitability of, 110
Dilator mechanism, tonus of, 259
Direct sensory cerebellar tract of Edinger, confusion created by term, 42
Disorientation by lapse of vision, 297
Distant vision, fixation for, 252
Disturbances in equilibrium, subjective interpretation of, 241
Dog, ocular movements in the, 319
Dogs, Ferrier's experiments on, 292, 293
  stimulation of cerebellum of, 291
Dorsal accessory olivary nucleus, 33
Dorsal decubitus, method of, in counteracting nausea in rotation sickness, 190
Dorsal fasciculi, constituents of, 79
  pathways of, 82
Dorsal-spinal-ganglion cells, emigration of, 22
Dorsal spinal root ganglia, inflammation of, 31, 35

Dorsal spino-cerebellar fasciculus, 43
  pathway of, 81, 82
Dorsal spino-cerebellar tract, origin of, 81, 82
Drugs, effect of, upon the circulation in rotation sickness, 190
  the influence of, upon the circulatory changes that occur with rotations, 134–139
Ductus endolymphaticus, 4, 6, 7
Dyspepsias, nervous, origin of, 167

Ear, diagram of the perilymphatic and endolymphatic spaces of the inner, 8
  internal, measurement of the, 3
  outer wall of, 3
  vestibule of, 3
Early metencephalon, dorsal zones of, 48
Edinger-Westphal nucleus, 23
Effector neurone, common path of, 119
Efferent cerebellar impulses, distribution of, 299
Efferent cerebellar path, through inferior olives, 55
Efferent cerebellar paths, ocular, 27
Efferent cerebellar tracts through undefined paths, 96
Efferent cerebello-pontine fibres, end of, 60
Efferent cerebello-pontine tract in the middle peduncle, fibres of, 60
Efferent fibres from the optic thalamus, 64
Efferent paths, 45, 47, 51, 53, 54, 55, 56, 60, 61, 63, 67, 68, 72, 74
Eighth nerve, fibres of, 13
Electrotonus, phenomena of, 279
Eleventh nerve, bulbar or accessory portion of, 34
Emboliformis, part of nucleus dentatus, 58
Embryological studies of Kölliker, 40
Eminentia arcuata, 4
Eminentia teres, 27
End-brain, constituents of, 69
Endolymph, current of, 266, 284, 286
  flow of, 267, 274
  forcing the, 266, 278
  the, 6
Epicritic fibres, description of, 94
Equilibration, active, 219
  adjustments of, 312
  disorders of, 181
  effects of aural irrigation upon, 237–249
  reflex acts of, 313
Equilibrium, disorders of, 243
  disturbances of, 117, 127, 130, 270
  disturbances of, compensated by hypnotic suggestion, 218
  effect of rotation upon, 192–197
  maintenance of, 298
  subjective interpretation of disturbances in, 241
Erysipelas, a cause of nystagmus, 333
Ewald test meal, description of, 164
Extensor muscles, tonus of, 308
External auditory canal, irrigations of, 20
  nystagmic movements in irrigation of, 20
External geniculate body, fibres from the, 70
External rectus muscle, 27
Eye, flexible mobility of, 302
  long ciliary nerves of, 108
  movements of the, 20

Eye area, removal of part of the frontal, 326
Eye movements, mechanisms controlling horizontal, 299
  in response to vestibular stimulation, 41
Eye muscles, fibres for movements of, 76
  nuclei of the, 15
Eyeball, antero-posterior axis of the, and rotary nystagmus, 199
  centre of rotation of, 203
  muscular apparatus of, 203
Eyeballs, movements of the, 320
  protrusion of, 292
Eyes, adjustments of the, 327
  conjugate movements of the, 48
  conjugate deviation of, 288, 317
  direct movements of, 316
  effects of rotation upon the, 198–206
  exclusion of the lateral movements of, 240
  fibres from the, 95
  jumping of the, 216
  lateral movements of, 315
  limited rotation of, 315
  movements of the, 20
  movements of, in response to loud sound, 15
  nystagmic movements of the, 205
  rotation of, 271
  study of deviation of, 286
  turning of the, 313
  voluntary control of rotary movements of, 240

Facial muscles, motor innervation of, 31
Facial nerve, motor and sensory root of, 12
  nucleus of, 25
Fasciculus, definition of a, 79
Fasciculus retroflexus of Meynert, origin of, 65
Fasciculus solitarius, fibres of, 32, 33
Fasciculus sulco-marginalis, 51
Fastigio-bulbar fibres, 58
Ferrier's experiments on cerebello-pontine fibres, 45
Fibre paths, in red nucleus, 311
Fibres of the cerebellum, 45–49
  of the corpus striatum, 73
  of the corpus subthalamicum, 67
  of the inferior colliculi, 53
  of the inferior olivary nucleus, 54
  of the lateral fillet, 52
  of the lateral geniculate body, 52
  of the mammillary nucleus, 68
  of the median geniculate bodies, 54
  of the mesial fillet, 49
  of the optic thalamus, 62
  of the pontine nucleus, 59
  of the red nucleus, 60
  of the rhinencephalon, 72
  of the substantia nigra, 67
  of the superior colliculi, 51
  of the superior olivary nucleus, 56
  of the vestibular nerves, 40–43
  of the worm, 58
Fifth nerve, a mixed nerve, 28
  chief motor nucleus of, 28
  connections of motor part of, 29
  connections of sensory part of, 28
  fibres of, 28
  motor fibres of, 29
  sensory nucleus of, 28
Fila anastomica, 12
Fishes, vestibular nerve of, 20, 71
  optic fibres of, 21
Flechsig, tract of, 43

Flocculus, irritation of the, 292, 301
Food, active absorption of, 284
  improperly digested, 167
Foramen of Magendie, situation of, 59
Foramina nervosa, 13
Foramina of Luschka, 59
Forel, decussation of, 61
  field of, 67
Formatio reticularis, periphery of lateral field of, 41
Formatio reticularis grisea, cells of, 44
  traversed by sensory root, 32
Fossa sulciformis, position of the, 3
Fourth nucleus, position of, 26
Fovea vagi, 32
Frenulum veli, 27
Frog, decerebrate, jumping of, 54
Frogs, section of the horizontal canals of, 126, 127
Frontalis muscles, 31
Fronto-cerebellar tract, constituents of, 59
Fundic musculature, contraction of, 180
Fundus oculi, changes that occur in the, during rotations, 206

Galvanic current, effects of, upon the semicircular canals, 268-272, 279
Galvanic stimulation of the cervical sympathetic, 109
Galvanism, mechanism of, 288
Galvanization of the sympathetic in the neck, 261
Ganglion trunci vagi, homologues of, 36
Ganglionectomy, general effect of, 109
Gasserian ganglion, contents of, 28
  inflammations of, 30
  removal of, 30
Gastric congestion in aural irrigations, 177
Gastric crises of tabetic origin, irrigations of little effect in, 246
Gastric disturbances, origin of, 182
Gastric glands, secretory fibres for, 101
Gastric juice, secretion of normal, 172
Gastric motility, testing, 234
Gastric mucous membrane, congestion of, 181
  secretion of fluid by, 175
Gastrin, 171
Geniculate ganglion, inflammation of, 30
  situation of, 29
Gennari, the white line of, 78
Glasserian fissure, 30
Globosus, part of nucleus dentatus, 58
Globus pallidus, 67
Glosso-pharyngeal nerve, stimulation of the, 166
Golgi method, use of, 79
Golgi-Mazzini organs, structure of, 85
Goll, fasciculus of, 82
Gowers' tract, description of, 83
  fibres from, 46
Grey matter, cells of, 86
Grey matter of the cerebellum, 57, 58
Grey rami, 100
Grey root of the optic nerves, 69
Gudden, fibres of inferior commissure of, 54
Gyrus fornicatus, anterior portion of, 78

Habenulo-peduncular tract, fibres of, 65
Hair cells, cylindrical, description of, 10
Head, deviations of, in rotations, 277, 278

Head, fibres for glands, blood-vessels, and plain muscle of the, 100
  ganglia of, 98
  turning of the, 313
Head movement, centres for, 313
Heart, accelerator fibres for, 102
Helweg, fasciculus of, 82, 83
  fibres from, 55
Helweg's bundle, connection of, 42
Hemisphærium, the, 69
Herpes zoster, origin of, 31
  of the tympanum, 30
Hippus, the study of, 250, 251
Homolateral pupillary constriction, 261
Horizontal canal, openings of the, 4
Horizontal nystagmus, 195, 278, 295
  movements of, 195, 199, 200, 242, 300, 301
Hot irrigations, effects of, upon the left ear, 237
Human cerebellum, development of, 48
Hydrochloric acid, effect of, upon gastric stasis, 174
Hyoscyamin, effect of, in rotation, 137, 138
  use of, in counteracting effects of rotation sickness, 191
Hypnosis, before, during, and after irrigations, 212–218
  waking, 184, 213
Hypnotic state, after-effects of, 185
Hypnotic suggestion, as a means of counteracting irrigation sickness, 219
  disturbances of equilibrium following irrigations compensated by, 218
  notes taken of the effect of, in aural irrigations, 183, 212
Hypnotism as a curative agent in all sorts of conditions, 188
Hypoglossal nerve (the twelfth), a pure motor nerve, 35

Impulses, inhibitory, paths of, in cord, 92
  from semicircular canals, 92
  in spinal cord, paths of, 92
  mutual reinforcement of, 177
  reflex, paths of, in cord, 92
  sensory paths of, in cord, 92
Inferior cerebellar commissure, bundles of, 58
Inferior colliculi, connections of the, 53
  efferent fibres of, 53
Inferior colliculus, cochlear paths to the, 56
  neurones of the, 16
Inferior frontal convolution, cortex of, 26
Inferior medullary velum, white matter of, 59
Inferior olivary nucleus, connections of the, 54–56
Inferior olive, fibres from, 55
  lesions of, 42
  relation of, 44
Inferior peduncle, efferent cerebellar tracts by way of the, 96
  paths through the, 94, 95
  section of, 314
  study of, 314
Inhibition, intimate nature of, 122
  period of, 300
  seat of, 122
  state of, 294, 295
Inhibitory impulses, 92
Internal auditory artery, 37
Internal auditory vein, blood returned by, 37
Internal cerebellar nuclei, 57
Internal rectus, inhibition of, 110
Interpeduncular ganglion, 66

Intersegmental tracts, meaning of, 90
Intestine, rhythmic segmentation of, 168
Intestines, rhythmic contractions of, 166
Invertebrates, sensory cells of, 22
Irrigating fluid, temperature of, 242, 243, 248
Irrigation, balancing tests before, 213
  equivalents of, 247-249
  onset of, 240
Irrigation tests, 270
Irrigations, aural, the general effects of, 207-219
  cold, effect of, 233
  cold unilateral, 235
  cold bilateral, 235
  experiments with cool, 243
  general study of, 251
  immediate effect of hot and cold, 254
  of the left ear, 239
  walking after, 244

Jugular foramen, and ganglion of vagus, 33
Jugular vein, blood and lymph passing into from labyrinth, 38

Katelectrotonus, state of, 280
K I test for absorption, 186-189, 236
Knee-jerks, deviations of, from the normal, 245

Labium tympanicum, 13
Labyrinth, anatomy of, 3-11
  blood-supply of the, 37-39
  bony, diagram of, 5
  bony, division of, 3, 5
  vessels of, 38
Labyrinthine artery, part of brain system, 37
Labyrinthine balanced mechanisms, 294
Labyrinthine impressions, associated with cerebellar function, 179
Labyrinthine impressions, corroboration of, 277
Labyrinthine nystagmus, 296
Labyrinthine paths, 71
Labyrinthine phenomena, mechanism of, 274
Labyrinthine receptors, effect of cold upon, 181
  effect of hot irrigations on, 272
  effect of rotations, aural irrigations, and galvanism on, 273-287
  effects of thermic irritation of, 219
  irritation of the, 172, 248
  irritation and irregular stimulation of, 181
Labyrinthine tonus mechanisms, existence of bilateral, 125
Lagena of lower animals, 11
Lamina cinerea, description of, 69
Lamina spiralis ossea, position of, 4
Laminæ, secondary and tertiary of cerebellum, 57
Langley, post-ganglionic fibres of, 90
Larynx, extrinsic muscles of, 35
Lateral cochlear nucleus, 14
Lateral fillet, connections of the, 52, 53
  fibres from nucleus of, 53
Lateral geniculate body, connections of the, 52
  grey nuclei of, 62
Lateral horn cells, analogue of, 44
Lateral occipital lobe, lesions of, 78
Lateral root, fibres of, in man, 70
Lateral ventricle, inferior horn of, 75
Lateral vestibular nucleus, 18
Legs, sleepy feeling in, 244
Lemniscus, connections of the, 49, 50
Lemniscus lateralis, 52, 53

Lenticular nuclei, fibres of, 63
Levatores palpebrarum, innervation of, 31
Ligamenti labyrinthi canaliculorum, 7
Lingula, rudimentary folia of, 59
Lissauer, constituents of zone of, 80
Locomotion, reflex mechanism of, 91
Locomotor ataxia, effect of irrigations in, 246
Lump-sensation in the stomach, 268
Lump-sensations during rotations, 170
Luschka, foramina of, 59
Luys, position of nucleus of, 67

Maculæ acusticæ, measurements of, 9
Maculæ of the utricle, hairs of, 11
Magendie, foramen of, situation of, 59
Mammifera, otoliths in, 128
Mammillary nucleus, connections of the, 68–72
  fibres to the, 68
Mammillo-tegmental tract, fibres of, 69
Mammillo-thalamic tract, 68
  fibres in, 63
Massa intermedia, constituents of, 62
Mechanisms, balanced, 329
Median geniculate bodies, fibres from, 54
Median geniculate bodies, connections of the, 54
Median geniculate body, neurones of the, 16
Median sagittal bundle of superior medullary velum, 59
Median vestibular nucleus, 18
Medulla, blood-supply by vertebral arteries, 38
Medulla oblongata, 27
  blood-supply of, 38
Medullary circulation, nature's defence against failing, 182
Medullary fibres, degeneration of, 107
Medullary lamina, fibres of, 62
Meissner's corpuscles, development of, 84
Meissner's plexuses, cells and fibres of, 165
Meissner and Auerbach, plexuses of, 98
Membranous canals, experiments of Ewald and Breuer on, 273
Membranous labyrinth, position of, 6
Memory, loss of, 78
  of printed words, centre for, 78
Mesencephalic centres in fishes, 20
Mesencephalic root, descending, 29
Mesencephalon, in fishes, 178
  in lower animals, 71
Mesenteric nerves, stimulation of, 173
Mesial fillet, connections of the, 49, 50
Meynert, commissure of, 50
  fasciculus retroflexus of, origin of, 65
Middle cerebellar peduncle, contents of, 46
Middle peduncle, efferent cerebellar tracts through the, 96
  paths through the, 95
Migraine and the cerebellar mechanisms, 336
Modiolus, central canal of the, 13
Monakow's tract, fibres of, 86
Monkey, pupil constriction in the, from the angular gyrus, 113
  stimulation of various parts of cerebellum of the, 290
Monkeys, division of the internal rectus of, 316

Morphin, effects of, in rotations, 136
  nausea caused by, 178
Motility, tests for, 189
Motor nerves, influence of auditory impulses on, 15
Motor neurones, influence of psychic impressions over, 179
Motor writing centre, existence of, 78
Mucous membrane, decoctions and extracts of the pyloric, 171
  effect of extracts made from various portions of, 186
Mucus, excessive amounts of, 176
Muscle spindles, fibres of, 85
Muscles of the body, tonus of, 93
Muscles of the face, motor innervation of, 31
Muscles of the pharynx, motor fibres of, 32
Muscular apparatus of the eyeball, 203
Muscular contractions, jerky irregularities in, 247
Myelin sheath, first appearance of, 72

Nausea and vomiting, mechanism of, 132
  phenomena of, 177, 182
Nausea, caused by morphin, 178
  origin of feeling of, 178
Near vision, fixation for, 252, 255
Nerve centres, rhythmic activity of, 257
Nerve deafness, 246
  aural irrigations a possible aid in the treatment of, 249
Nerve degeneration, danger of, 247
Nerve elements, degeneration of, 110
Nerve fibres, origin of, 13
Nerve roots, T-bifurcation of the posterior, 80
Nerve, the eighth, description of, 12
Nervous dyspepsias, origin of, 167
Nervous system, intimate functional relations of the various parts of, 122
Nervus erigens, sympathetic fibres of, 102
Neuro-epithelial cells, 13
Neuro-epithelium, intimate relation of nerve filaments to, 17
Neuro-vascular mechanisms, exhaustion of the, 136
Ninth nerve, motor portion of, 32
  root fibres of, 33
Nitroglycerin, effect of, in rotations, 135
Nociceptive reflexes, 121
Nociceptors, definition of, 120
Nose and mouth, nerves for glands and blood-vessels of mucous membrane of, 101
Nucleo-cerebellar tract, constituents of, 19
Nucleus ambiguus, fibres from, 35
  position of, 32
Nucleus cuneatus, fibres from, 49
Nucleus dentatus, fibres of, 43
  atrophy of, 43
Nucleus fastigii, axones of, 58
  end of, 44
Nucleus gracilis, fibres from, 49
Nucleus lateralis, cells of, 44
Nucleus lemnisci lateralis, 15
Nucleus masticatorius, 28
Nucleus olivarius superior, 14
Nucleus pontis, fibres from, 57
Nucleus ruber, connections of the, 60–62
Nucleus Tegmenti, connections of the, 60–62
Nucleus trapezoideus, cells of, 56

Nucleus trapezoideus, constituents of, 14
Nucleus ventralis, position of, 32
Nystagmus, apparent movement of objects in horizontal, 200
  cause of, in aural irrigations, 243, 295
  cerebellar differentiated from labyrinthine, 334
  due to rotation, 197
  from erysipelas over mastoid area, 333
  general effect of a mixed horizontal and rotary, 263
  horizontal, 195, 278, 295
  in animals, 329
  in rotations, coordinated movements of, 304
  labyrinthine, 296
  latent vertical, 239
  miner's, 332
  mixed, 271, 274
  momentary, 38, 39
  nervous mechanism of, 289
  occurrence of, 331-335
  ocular, 296
  of rotations, mechanism of, 288-290
  physiological, 329
  rotary element of, 195, 200
  secondary, 203
  simultaneous presence of three forms of, 240
  spontaneous, 320, 327, 329
  teething, 333
  twofold, 197
  use of term explained, 198
  vertical, 204, 244, 266, 267, 276, 282, 300, 321
  violent, 215
  visible, 332

Oblique, paresis of right superior and left inferior, 306
Occipital cortex, 70
  fibres of, 52, 63
Ocular area, stimulation of the frontal, 303
Ocular centre, blood-supply of, 39
Ocular and cerebellar centres, blood-supply of, 38
Ocular movements and nystagmus, 315-330
Ocular movements, association of, 113
  location of centres for reflex, 260
Ocular muscles, associations of, 285
  innervation of, 39
  summary of the relations of, 284-287
Ocular nystagmus, 296
Oculo-motor centres, 20
Oculo-motor fibres, origin of, 24
  decussation of, 24
Oculo-motor nuclei, relations of the semicircular canals to the, 306
Olfactory cortex, projection fibres of, 71
Olfactory lobe, fibres of, 75
Olfactory nerve, central relations of, 22
  primary afferent neurones of, 83
Olfactory organs, recession of, 179
Olivary cells, axones of, 55
Olivary fasciculus, connection of, 42
Olive, experimental phenomena of, 44
Olivo-cerebellar fibres, 43
Optic and sensory spinal elements, analogy between, 22
Optic atrophy, 296
Optic chiasm, location of, 69
  paths of, 69
Optic nerve, components of, 22
Optic nerves, grey root of, 69
Optic pathways, 71
Optic thalamus, connections of the, 62-65
  fibres from cells of, 50
  fibres to the, 61

Optic tract, paths in the, 70
Optics, principles of, 202, 203
Orbicularis palpebrarum, 31
Organism, defences of the, against noxious substances, 167
Organs of special sense, fibres from, 96
Otoconium, composition of, 11
Otolithic apparatus, inactive state of, 134
Otolithic membrane, description of, 10
Otoliths, composition of, 10
  in mammifera, 128
  of fishes, 11

Pain, sensation of, paths for, 83
Pancreatic glands, secretory fibres for, 101
Paradoxical pupil contraction, phenomenon of, 107
Paradoxical pupil dilatation, modus operandi of, 258
Paræsthesiæ, occurrence of, 245, 335
Pars intermedia, 12
Passive rotation, effects of, 130–139
  disturbances in, 130
  position of the body during, 130
Peduncle, inferior cerebellar, 14
Pedunculus corporis mammillaris, fibres of, 69
Peripheral neurones, location of afferent, 83
Peripheral terminals of efferent paths, 94
Peripheral terminations of afferent paths, 83–94
Peristalsis, after vagus section, etc., 168
  first signs of, after vagus section, etc., 169
Peripheral stump of vagus and splanchnic nerves, stimulation of, 165
Pharynx, innervation of muscles of the, 32
Pigeons, experiments on the semicircular canals of, 125, 126, 127
  Ferrier's experiments on, 293
  pupil constriction in, 113
Pig's stomach, cardiac and pyloric sphincters of, 166
Pilocarpin, in inflammatory conditions affecting the internal ear, 212
Planum semilunatum, 9, 10
Pons, supplied by vertebral arteries, 38
Pontine nucleus, connections of the, 59, 60
  fibres from cells of, 45
Posterior commissure, paths of, 66
  constituents of, 66
  position of, 58
Posterior canal, ampullary end of, 4
Posterior longitudinal fasciculus, connections of, 51
  crossed and uncrossed fibres to the, 41
  fibres of, 26, 66, 69
  relation of, to nerves, 41
Posterior median fasciculus, 41
Posterior median sulcus, area of, 79
Posterior nerve-root, collaterals and terminals of, 80, 81
Posterior nerve-roots, T-bifurcation of, 80
Posture, tonic reflexes of, 122
Potassium, the effect of bromid of, in rotation, 136
Præcentral gyrus, 32
Preganglionic fibres, 100
  course of, 100
  of the sympathetic nervous system, 90, 99
Prepotent reflexes, 121
Primary sensory neurones, cells of, 32
Proprio-spinal neurone, connections of, 119

Protocols of experiments on rotation subjects, 139-163
Protocols on irrigation subjects, 220-232
Pseudo-affective reflex, 121
Psychical secretion, gastric, 171
Pulse-rate, changes in, during irrigations, 253
Pulvinar, fibres from the, 70
Pupil constriction, centre for, 107
  in the monkey and in pigeons, 113
Pupil dilatation and constriction, effects of, 111, 113
Pupil, rhythmic dilatations of the, 259
Pupillary constriction, homolateral, 109
  mechanism of, 105, 106
Pupillary dilatation, mechanisms of, 251, 252
Pupillary fibres, views on the paths of the, 106
Pupillary movements, the paths involved in, 105-113
Pupillary phenomena in hot irrigations, 250
Pupillary reactions, study of, 251
Pupillo-constrictor mechanism, hypertonus of, 258
Pupillo-constrictor path, 107
Pupillo-dilator muscle, existence of, 108
Pupillo-dilator tract, position of, 108
Pupils, condition of, following cold irrigations, 250
  inequality of, 260
  irregularity of, 107
  state of, in irrigations, 259
Purkinje cells, axones of, 45
  fibres from, 45
Putamen, fibres from the, 67
Pyloric circular fibres, inhibition of the, 166
Pyloric closure during rotations, maintenance of, 170
Pylorus, closure of, in rotations, 165
  delicate local mechanisms of, 170
  explanation of mechanism of opening of the, 166
  stenosis of the, and the labyrintho-cerebellar mechanisms, 336
  tightly closed in aural irrigations, 235
Pyramidal tract, lesion of direct, 86

Quadriceps extensor, inhibition of the, 296
Quadrigemina, inferior colliculus of the, 16

Rabbits, Ferrier's experiments on, 292
  section of the semicircular canals in, 126
  stimulation of cerebellum of, 290
Radial pulselessness in rotations, 131
Radix cochlearis, 12
Radix vestibularis, 12
Receptor organs, division of, 120
Recessus ellipticus, grooves of, 3, 6
Recessus utriculi, blind sac of, 6
Reciprocal innervation, 294, 297
  phenomenon of, 122
  theory of, 258
Rectus, paresis of the left external, 306
Recumbency, supine, potency of, in relaxing the pylorus, 186
Red nucleus, connections of the, 60-62
Red nucleus cells in animals and man, 65

Red nucleus neurones, axones of, 47
Reflex balanced mechanisms, disturbance of, 278
Reflex complication, 121
Reflex impulses, 92
Reflex paths, protective, 70
Reflexes, prepotent, 121
  compounding of, 121
  pseudo-affective, 121
Refraction, changes in, 256
  errors of, 257
Regurgitations in animals, 180
Retardation, reaction of, 267
Retina, movement of images upon, 262, 267
Retinal anæmia, 109
Retinal impressions of passing objects, 71
Retinal pigmented epithelium, 109
Retinal vessels, hyperæmia of, 261
Rhinencephalon, connections of the, 72, 73
  constituents of, 71
Rolando, cells in the substance of, 80, 81
  fissure of, 75
Roof nuclei, decussation of, 59
Rotary nystagmus, 267, 282, 283, 301, 322
  apparent movement of objects in, 201, 202
  mechanism of, 285
  production of, 195
  study of, 203
  vision impossible during, 262
Rotation, brisk, in the coronal plane, 195
Rotation, coronal element in, 199
  disturbances in, 130
  effect of acceleration upon subjects, 193
  effect of glycerin in, 135
  effect of mild, upon the circulation, 131
  effect of, upon equilibrium, 192–197
Rotation, effects of, upon the digestive apparatus, 164–182
  effects of, upon the eyes, 198–206
  effects of passive, 130–139
  increased blood-pressure in, 132
  in perverting gastric function, efficiency of, 234
  in the sagittal plane, 279
  nystagmus due to, 197
  position of the body during, 130
  protocols of experiments of, upon various subjects, 139–163
Rotation sickness, eructations in, 180
  hypnotic suggestion in offsetting effects of, 190
  prevention of, 183
  train of symptoms set up in, 138
  vascular congestion of stomach accompanying, 170
Rotation subjects, effect of swings upon, 192
  experiments on, 184–191
Rotation test meals, 164
Rotations, closure of pylorus in, 165
  diminished secretion of gastric juice in, 172
  displacement of the head in, 193
  effect of, in the coronal plane, 194, 203
  effect of, in the mesial plane, 194
  effect of repeated, upon the pulse, 132
  effects of strychnin and morphin in, 136
  in recumbency, 134
  in the sagittal and coronal planes, 132
  in the sagittal and diagonal planes, effects of, 196

Rotations, in the upright position, conclusions on, 133
    increase of salivary flow in, 189
    influence of drugs upon the circulatory changes that occur with, 134–139
    rise of blood-pressure after, 132
    salivary flow in, 164
Rotations with subject lying horizontally, 164, 165
Rotations with subject sitting upright, 164
Rubro-spinal tract, efferent impulses of, 61
    fibres of, 86

Saccule, hairs of, 11
    structure of, 9
Sagittal plane, effects of rotations in the, 196, 204
Salivary flow, increase of, in rotations, 164, 189
Salivary glands, secretory and vaso-dilator fibres for, 31
Salol test for stomach motility, 234
Salol tests in rotations, 164
Schultze, "comma" tract of, 89
Scratch reflex, end effect of positive sign, 121
Secondary nystagmus, 203
Secretin, gastric, 171
Secretin in the pyloric portion of the mucous membrane, 172
Secretogogues in food, action of, 171
Secretory fibres for salivary glands, 31
Semicircular canal apparatus, theories concerning, 128
Semicircular canals, blood-supply from internal auditory artery, 37
    connections of, 294
    description of, 4
    effects of the galvanic current upon the, 268–272, 279
Semicircular canals, impulses from, 92
    intimate relations between and the function of equilibration, 125
    literature of, 125
    location and direction of the, 266
    membranous, 7
    nausea and vomiting following disturbances of, 177
    not in direct relation with the medullary vaso-constrictor centres, 133
    of pigeons, 125
    of rabbits, 126
    of frogs, 126
    openings of, 6
    peripheral end organs of the, 117
    physiology of the, from the standpoint of animal experimentation, 125–129
    receptors of, 280
    relations of the, to the oculo-motor nuclei, 306
    structure of, 9
    study of, 114, 125, 289
Semilunar lobule, irritation of, 292
Senile deafness, cause of, 37
    hot irrigations in, 246
Sense of seeing, hearing, and smelling, 78
Sense of taste, cells related to, 32
Sensory cerebellar tract, 42
Sensory cranial nerves, fibres from reception nuclei of, 49
Sensory fibres, systems of, 93
Sensory impulses, 92
    question of, 85
Sensory nerves of external auditory canal, effects of thermic stimulation of, 251
Sensory neurones, peripheral endings of, 84
Sensory pupil reflex, 111

Septo-marginal tract, fibres of, 89
Septum transversum, 8
Seventh nerve, afferent fibres of, 30
  motor nucleus of the, 31
Sheep, section of right vestibular nerve in, 319
Shock, Porter's view of the mechanism of, 134
  Crile's view, 134
  Henderson's view, 134
Simple reflex arc, composition of, 118
Sinus posterior, connection of, 6
Sinus superior, position of, 7
Sinus utricularis, wall of the, 7
Sixth nerve, fibres to nucleus of, 56
Sixth nucleus, fibres to the, 40
  multipolar cells of, 27
Skin, temperature of the human, 207
Skin-vessels, constriction of, 275
Smell, centre for, 78
  perversion of sense of, 179
Sound vibrations, the tympanum as a means of communication of, 246
Spatium perilymphaticum, 6
Spinal cord, ascending tracts of, 79
  fibres to the, 41
  impulses, paths of, in, 92
  pathways in the, 79–83
Spinal dog, a, 91
Spinal ganglia, fibres from, 84
Spinal ganglion, cells of the, 13
Spinal nerves, root cells of the, 19
Spinal reflexes, pathway for, 80
Spinal segment, somatic and splanchnic roots of, 35
Spinal vestibular nucleus, 18
Spino-spinal neurones, 91
Spino-spinal paths, existence of reflex, 91
Spino-tectal and spino-thalamic tracts, cells of, 81, 82
Spino-tectal fibres, 51
Spiral canal, dendritic processes in the, 13
Splanchnic nerve, effect of stimulation of, 166
Splanchnics, bilateral section of the, 168
  section of the, 169
Spontaneous nystagmus, 320, 327, 329
Staloliths, function of, 11
Stasis of the stomach contents in rotations, 164
Statocysts, function of, 11
Stomach, condition of the, in the hypnotic state, 184
  effect of rotation upon, 176
  loss of muscle tonus in, 165
  lump-sensation in, 180
  resistance of the, to all sorts of insult, 167
Stomach and intestine, general contractions of, 166
Stomach cavity, division of, into compartments, 178
Stomach functions, effect of aural irrigations upon the, 283
Stomach motility, salol test for, 234
Stomach movements, inhibition of, through the splanchnics, 169
  initial signs of, after vagus section, etc., 168
Stomach muscles, augmentor fibres for, 165
Stomach tube, hemorrhage attributed to, 177
Stomachs, experience in washing out, 173
Stooping, sudden, and vertigo, 38
Stratum zonale, fibres of, 63
Striæ acusticæ, 14, 27
Striæ medullares, fibres of, 65
  probable constituents of the, 65–67

Strychnin and atropin, influence of, in rotations, 184
Subarachnoid space, communication of, with fourth ventricle, 59
Sublingual ganglion, named by Langley, 31
Sublingual salivary glands, secretory and vaso-dilator fibres of, 31
Submaxillary ganglion, cells of the, 31
Submaxillary glands, secretory and vaso-dilator fibres of, 31
Subordinate nystagmus, 197
Substantia ferruginea, pigmented cells of, 29
Substantia gelatinosa, 28, 33
  fibres from, 50
  neurones within the, 19
Substantia nigra, fibres from, 51, 52
  location of upper extremity of, 67
  probable connections of the, 67
Subthalamic body, grey nuclei of, 62
Subthalamic region, position of, 66
"Successive degeneration," method of, 92
Summer warmth, relaxing effects of, 181
Superficial sensibility, 93
Superior cerebellar commissure, fibres of, 58
Superior cerebellar peduncle, paths of, 61
Superior colliculi, fibres to, 70
Superior colliculi of the quadrigemina, connections of, 51, 52
Superior medullary velum, 27, 59
Superior olivary nucleus, 14
  connections of the, 56–58
  fibres from, 52, 53
Superior olive, fibres to the, 41
Superior orbital fissure, 27
Superior peduncle, efferent cerebellar tracts through the, 96
  paths through the, 95
  section of, 313
Superior vestibular nucleus, 18
Suprasegmental tracts, meaning of, 90
Sustentacular cells, description of, 9
Swing, experiments with, 278
Swings, effect of, upon rotation subjects, 192
Sylvius, aqueduct of, 22, 23, 24, 29, 48, 50, 66, 69, 178
  grey matter of, 69
Sympathectomy, general effect of, 109
Sympathetic autonomics, 32
Sympathetic ganglionectomy, 110
Sympathetic nervous system, preganglionic fibres of, 90, 99
Sympathetic or autonomic nervous system, 98–104
Sympathetic reflexes in the normal individual, 99
Sympathetic system, normal mode of stimulation of, 103
Synkineses, 25
Synopsis of anatomical connections, 40–77

Tactile sensation, pathway for, 82
Tænia semicircularis, 75
Tangoceptive receptors, 121
Taste, loss of, 30
  sensations of, 30
  sense of, 32
Taste buds of tongue, cells of, 84
Taste fibres of the chorda tympani, 30
Technical terms, explanation of some confusing, 117
Tecto-bulbar tracts, fibres of, 51

Tecto-spinal tract, 51
  fibres of, 86
Tegmentum of the pons, arcuate fibres of the, 40
Telencephalon, constituents of, 69
Temperature, paths for sensation of, 83
Temperature of the human skin, and thermic reactions, 207
Temporo - occipito - cerebellar tract, constituents of, 59
Tenth nerve, cell bodies of, 33
  efferent fibres of, 34
  motor fibres of, 34
Test meals, nature of, 235
  with rotations, 164
Thalamus, fibres to the, 70
  ventral surface of, 62
Thalamo-cortical tracts, 75
Thalamo-mammillary tract, fibres of, 65
Third nerve, fibres of, 107
  nucleus of, 22
Third nucleus, grouping of nerve-cells of, 23, 24
  region of, 38
  relations of, to the seventh, 26
  connections of, 25
Thomas, fibres of tract of, 89
Tobacco-smoke, nausea excited by odour of, 138
Tongue, anterior two-thirds of, and the sense of taste, 30
  intrinsic muscles of, 85
  muscles of the, 35
  taste buds of, 84
Tonic reflexes of posture, 122
Touch cells, nerve fibres of 84
Tonus innervation, 278
Toxemia of the cerebral centres, 332
Tract, a physiological term, 79
Tractus cerebellaris acusticus, 42
Tractus spiralis foraminosus, 13
Tractus strio-thalamicus, fibres of, 60, 63
Tractus vestibulo-spinalis of Monakow, 42
Trigonum habenulæ, 65
Tuber cinereum, 69
  grey matter of, 71
Tuberculum acusticum, 14
  cells of, 15
Twelfth nerve (hypoglossal) supplying muscles of the tongue, 35
Tympanum, herpes zoster of the, 30
  inflammatory exudations in the, 246

Uncinate fasciculus, connection of, 74
Unilateral irrigation, 276
Utricle, divisions of, 6
  macula acustica of, 7
  structure of, 9
Utriculus proprius, division of, 6

Vago-splanchnic section, 169
Vagus, preganglionic fibres of, 101
  or pneumogastricus, 33
  secretory fibres contained in, 171
  stimulation of the gastric branches of, 170
Vagus centre, effect of irritation of, 167
Vagus centres, irritation of, from rotations, 170
Vagus irritation, initial effect of, 172
Vagus stimulation, by rotation, effects of, 135
  effects of central, 135
  through central stump, 165
Vascular changes, alternating, 260
Vaso-constrictor fibres, distribution of, 103
Vaso-dilator fibres, distribution of, 103
  for salivary glands, 31
Vaso-motor changes, extent of, 113

Vaso-motor mechanisms, tonic effects of cold upon, 181
Vater-Pacini corpuscles, 84
Ventral cochlear nucleus, 14
  extent and constitution of, 17
Ventral horn, grouping of cells in, 93
Ventral marginal fasciculus, 41, 44
Ventral spino-cerebellar tract, cells of, 81
Ventral stalk, fibres of, 64
Ventral zones, of metencephalon, 48
Vermiform process, irritation of the upper, 289
Vermis, electrical stimulation of the upper, 289
  vestibular root-fibres to, 57
Vertical nystagmus, 204, 244, 266, 267, 276, 282, 300, 321
Vertigo, externalization of, 199
  from sudden stooping, 38
Vestibular apparatus, afferent paths of the, 298
  crossed relation of, 309
Vestibular connections, discussion on, 48
Vestibular nerve, connections of the, 40–43
  division of, 13
  entrance of the, through the macula cribrosa, 5
  fibres of, 43
  indirect connection with parts of the vestibular nucleus, 18
  in fishes, 20, 71
  phenomena that follow section of, 285
  section of, 42
Vestibular nucleus, crossed and uncrossed fibres from, 48
Vestibular portion of eighth nerve, fibres of the, 17
Vestibularis, section of the, 309
Vestibulo-olivary tract, 42
Vestibulo-spinal tract, fibres of, 20
Vicq d'Azyr, bundle of, 68, 72
Vieussens, annulus of, 102, 108
Violent nystagmus, in aural irrigations under hypnosis, 215
Visible nystagmus, 332
Vision, disturbances of, on sudden stooping, 38, 39
  distant, fixation for and pupil dilatation, 252
  near, fixation for and pupil contraction, 252, 255
Visual acts and conceptualization, 78
Visual fibres, in animals, 69
  in man, 70
Visual organs of lower animals, 71
  representation in the occipital cortex, 71
Vomiting and nausea, interpretation of phenomena of, 177
Vomiting, bye-results of, 134
Vomiting mechanism, otolithic apparatus related to, 179

Waking hypnosis, 184, 213
Walking, after irrigations, 244
Wasting diseases, nystagmus and vertigo in, 39
Word-sounds, loss of memory of, 78
Worm, connections of the, 58, 59
Writing centre, existence of motor, 78

Zone of Lissauer, constituents of, 80

INDEX OF AUTHORS REFERRED TO IN THE SEMICIRCULAR CANALS

Adamük, 109
Alexander, 246
Anderson, 105, 106, 107, 109, 110, 111, 113, 258, 259
Apolant, 107
Argyll-Robertson, 106
Auer, 168, 169
Auerbach, 165, 174

Bach, 106
Baginsky, 125
Bailey, 90
Balogh, 111
Balthazard, 173
Bannister, 125
Bárány, 125
Bayliss, 98, 103, 166, 171, 186
Beard, 184, 261
v. Bechterew, 15, 24, 26, 43, 44, 45, 46, 47, 50, 51, 56, 57, 60, 67, 70, 95, 96, 105, 106, 111, 117, 125, 127, 259, 260, 298, 306, 308, 309, 310, 314, 321
Beevor, 77, 295, 313, 315, 327, 331
Bernard, 111
Bernheimer, 106
Biehl, 125, 127, 285, 291, 306 319, 324
Bogroff, 106
Braunstein, 111
Breuer, 125, 128, 265, 273
Brown, 25, 78
Brown-Séquard, 125
Brübaker, 166
Bruce, 19, 43
Bruck, 125
Brunton, 34
Budge, 105, 108, 167
Bumm, 107
Burdach, 79
Burton-Opitz, 34, 101, 170

Cajal, 79, 119
Campbell, 31
Campos, 109
Cannon, 167, 169, 173, 180
Charpy, 5
Chauveau, 111
Clarke, 11, 34
Collins, 107, 113
Comus, 257
Crile, 184
Crum-Brown, 125, 128
Cunningham, 42
Cushing, 30
Cyon, 102

Darkschewitsch, 106
Darwin, 125
De Cyon, 102, 125, 126, 128, 286
Deiters, 42, 298
Dejerine, 60, 73, 74
Delage, 125
Demtschenko, 109
Dogiel, 109
Doyon, 256
Dreyfus, 125
Dufour, 257
Duval, 25, 40, 288, 297

Edinger, 42, 49, 60, 62, 66, 68, 73, 74
Edkins, 171, 186
Engelmann, 125
Erlanger, 94
Ewald, 116, 125, 128, 265, 267, 273, 286

Ferrier, 15, 19, 25, 31, 40, 42, 45, 97, 106, 111, 112, 113, 114, 115, 116, 117, 167, 194, 260, 286, 289, 292, 293, 294, 302, 310, 313, 315, 317, 319, 320, 326, 327, 336
Flechsig, 77, 106
Flourens, 105, 125, 127
Forel, 67
François-Franck, 109, 112, 113
Fuchs, 195, 203, 263
Fuss, 109

Gaskell, 35
Goll, 79
Goltz, 103, 125, 127, 128
Gordinier, 40, 81, 95
Gowers, 83, 95
Gratiolet, 319, 324
Grunert, 109
Grutzner, 234
Gudden, 114

Haab, 111
Haberman, 246
Head, 31, 93
Heese, 109
Heine, 256
Henderson, 134
Henschen, 105
Hensen, 23, 110
Hess, 256
Hitzig, 111, 125
Hoche, 46, 82, 83
Högyes, 125, 286
Horsley, 77, 295, 313, 315, 327, 331
Howell, 92, 103, 108
Hunt, 30

Jackson, 125
James, 125
Jegerow, 107
Jessop, 258

Kahler, 24, 285
Katschew, 109
Klemoff, 42
Knoll, 106
Koenig, 125
Kölliker, 40, 42, 43, 55, 69, 82, 96, 178
Koryani, 125
Kreidl, 125
Kyle, 39

Laborde, 25, 40, 125, 288, 297
Lafarge, 313
Langendorff, 107, 109, 110
Langley, 31, 90, 94, 98, 99, 105, 107, 109, 110, 113, 169
Laslett, 80, 91
Lee, 125, 286
v. Lenhossék, 69
Lermoyez, 212
Leven, 319, 324
Levinsohn, 107, 109
Lewandowski, 110
Lodato, 109
Loeb, 21, 71, 125
Longet, 309, 313
Lucae, 125
Luciani, 115, 309, 317, 320, 324, 326, 329
Ludwig, 102
Lussana, 309, 313

Mach, 125, 128
Magendie, 314, 319, 324
Marchi, 97
Marina, 107
Maunoir, 108
May, 169
Meissner, 165, 174
Meltzer, 169
Mercier, 317
v. Monakow, 62, 91
Morat, 169, 256
Morris, 41, 42
Mott, 46, 81, 83, 115, 315
Munk, 25, 315
Murphy, 169

Neumann, 125
Nissl, 62

Obersteiner, 66, 68
Ollivier, 319
Onuf, 107, 113
Oppenheim, 78
Osler, 334

Panse, 310
Parfour du Petit, 109

Parsons, 21, 70, 71, 106, 108, 111, 112, 113, 257
Pawlow, 171
Perlia, 24, 285
Peterson, 334
Pick, 24, 285
Piersol, 11
Pitres, 113
Poirier, 5
Politzer, 212
Porter, 134

Quain, 23

Reid, Waymouth, 173, 258
Renzi, 320
Retzius, 11, 22
Risien-Russell, 40, 97, 115, 300, 303, 309, 315, 317, 319, 320, 321, 324, 326, 327, 329, 330, 332
Rivers, 93
Rockwell, 261
Rohmen, 257
Rolando, 314
Roosa, 261
Rosenberg, 109
Ross, 34
Rossolimo, 83
Roux, 173
Ruge, 106
Russell, 83, 317, 318
Rutherford, 170

Sabin, 18
Sacki, 106
Salkowski, 105
Salvioli, 173
Sappey, 5, 69
Saucerotte, 319
Scarpa, 125
Schäfer, 25, 40, 49, 78, 113, 115, 288, 298, 302, 315, 316
Schenk, 109
Schiff, 108, 309
Schmaus, 106
Schwartze, 125, 286
Selenkowski, 109
Sewall, 125
Shaeffer, 106
Shambaugh, 37
Sherrington, 80, 91, 92, 110, 112, 118, 119, 120, 121, 122, 259, 294, 295, 296, 300, 308, 315, 316, 318, 331
Siebenmann, 37
Sinitzen, 109
Solder, 82
Spamer, 125
Starling, 103, 166, 171, 172, 186
Starr, 24, 114, 285 333
von Stein, 125
Steiner, 125
Sylvius, 66, 69, 178

Techlenberg, 173
Terrien, 257
Tsuchida, 24, 25, 285
Turner, 19, 40, 42, 97, 106

Valentine, 167
Van Gehucten, 84
Van Giesen, 41
Verworn, 11
Vieussens, 102, 108
Volckers, 23, 110
Vulpian, 125

Waller, 105
Wanner, 125
Warner, 317
Weber, 110
Weir-Mitchell, 117, 320
Witmaack, 37
Wolferz, 109
Wolff, 106

INDEX TO SEASICKNESS

Abdominal blood, flow of, toward the heart in vomiting, 389
Abdominal organs, concussion of the, as a cause of seasickness, 494
Abdominal viscera, effects of protracted congestion of, in seasickness, 515
Absorption test, 348, 349, 351, 352, 365, 367
Absorption and motility tests, 347
Adaptation of the functional activities, the principle of, 382
Adrenalin, action of, 494
Afferent arcs, interference with, 497
Alcohol, action of, upon the cardio-vascular mechanism, 358
  effect of, upon the heart-structure, 359
  effects of, upon the circulation, 358
  relation of, to seasickness, 510
Amenorrhœa following seasickness, 513, 515
Ampullary receptors, active stimulation of, 364
  affected in mild forms of seasickness, 499
Analyses of specimens taken from seasick subjects, 342, 343, 350, 351, 353, 354, 360, 361, 366–368, 370, 371, 373–375, 377, 378, 380, 386, 387, 390–392, 394–396, 399, 400, 402, 403, 405, 406, 408, 409, 411–413, 415, 416
Angler, steamship, observations of seasick subjects on, 341, 343, 384, 385, 386, 387, 390–416, 421–430
Arteries, contraction of, in seasickness, 344
Astoria, steamship, observations of seasick subjects on, 346
Athletes, condition of, after sea voyage, 509, 510
  "warming up" of, 363
Atropin, effect of, on the cardiac vagus terminals, 391, 408
  especially valuable in combination with strychnin, 378, 384, 420
  value of, in warding off disagreeable effects of seasickness, 376, 420
Aural irrigations, rotations, and galvanism, insensitiveness to, 385

Balancing movements, well executed after strychnin and atropin at sea, 372
Barker's theory of seasickness, 479
Barnett's theory of seasickness, 484
Basche's sphygmomanometer, 482
Beard's theory of seasickness, 480
Berard's theory of seasickness, 479
Binz's theory of seasickness, 482

Books from ship's library, danger of infection from, 517
Broth, ordinary, efficient in seasickness, 420

Cabin, bad effects of a hot, stuffy, 382
  ventilation and temperature of the, in seasickness, 418
Caledonia, steamship, observations of seasick subjects on, 345–347, 432–453
Cardio-vascular mechanism, action of alcohol upon, 358, 359
Carsickness, labyrinthine defects in, 492
  the result of fear, 492
Cascara sagrada in seasickness, 348
Castalet, D'Ailhaud, theory of seasickness, 480
Cato the Elder on seasickness, 478
Cerebellar exhaustion in seasickness, 506
Cerebellum, exhausting effect of interference with, 504
Chapman's theory of seasickness, 479
Children immune from seasickness, 508
Circulation, changes in, at sea, 340
  disturbances of, as cause of seasickness, 498
  effect on, while travelling on a moving train, 365
  slow recovery of, after seasickness, 357
Circulatory mechanism, necessity for study of, in hydrotherapy, 383
Circulatory and gastric disturbances in seasickness, 487
Cocain, use of, contraindicated, 519
Cold air, good effect of, on the circulation, 381
Cold bathing, 418
  effects of, on the vasomotor mechanism, 352, 355
  tonic hyperemia after, 383
Colds at sea, 516
Communicable disease, danger of, in crowded cabins, 516
Concussion, gross, not the cause of seasickness, 493
Constipation, relief of, 522
Convalescents, fallacy of sending them to sea, 512
Cornelius's theory of seasickness, 482
Corning's treatment of seasickness, 483
Cygne, steamship, observations of seasick subjects on, 367, 368

Dancer, a professional, a victim of seasickness, 489
Darnall's theory of seasickness, 481
Darwin, Erasmus, theory of seasickness by, 478
Deaf-mutes, immunity of, to galvanism, 487
  immunity of, to rotation sickness, 503
Deaf persons immune from seasickness, 502
DeVries's theory of seasickness, 481
Digestion, phenomena of disturbed, 350
Digestive disturbances in seasickness, 344, 417, 418, 494
Disordered circulation, a secondary source of irritation in seasickness, 522
Dolor cerebri, indescribable sickening feeling, 360, 377, 388, 401

English Channel, its reputation for causing seasickness, 363

Equilibrium, reflex disturbances of, 497
Etiology of seasickness, 486–507
Ewald test meals, 343–414
Eye, a factor in equilibration, an important source of afferent impulses, 490
Eye disturbances, as a cause of seasickness, 491
Eyes, motions of the, a possible cause of seasickness, 505, 506
  motor mechanisms of the, 491
  relation of the, to the optic thalamus, 507

Features of seasickness, common, 347
Fehling's solution as a test, for sugar, etc., 352
Flasschoen's theory of seasickness, 484

Gaertner's tonometer, 482
Gastric contents, analysis of, 351, etc. See Analyses.
Gastric function, failure and perversion of, 504
Gastric juice, enhancing the flow of, 421
Gastric mucous membrane, decoctions of, an aid to absorption, 421
Gastric secretin inert in seasickness, 420
Gastric secretion, failure of normal, 406, 407
Gastric and circulatory disturbances in seasickness, 344
Gastro-intestinal motility, stimulation of, by atropin, 399
Guien's theory of seasickness, 479
Guillabert's theory of seasickness, 479
Gyroscope, principle of, suggested in preventing seasickness, 520, 524

Hagen-Torn's theory of seasickness, 483
Hazen's theory of seasickness, 481
Heart, strain on the, in seasickness, 512
Hints to seagoing travellers, 519
Hippocrates's allusion to seasickness, 478
History and literature of seasickness, 478–485
Homatropin, instillation of, 384, 385, 403
Horizontal nystagmus, cause of, 497
Hormones, a decided help in treating severe seasickness, 522
Hospital facilities on board ship generally poor, 515
Hot weather, effect of, on seasickness, 488
Hydrotherapy, cold procedures in, and the circulatory mechanisms, 383
Hypnotic suggestion, as a preventative of seasickness, 520
  effect of, on subject "F," 409
  not an efficient means of preventing seasickness, 420
Hypnotism, limited field of usefulness in seasickness, 520

Imagination, influence of, in the etiology of seasickness, 347
Immunes from seasickness, 509
Intermediate extract, 407, 420
Invalids being sent to sea, the fallacy of, 512
Irritation, testing prolonged effects of mild, in seasickness, 345

Jendrassik's reinforcement, 404

Jobert's theory of seasickness, 479

K I absorption tests, 342–409
Kinæsthetic sense, explanation of term, 506
Klein's theory of seasickness, 481
Koepke's theory of seasickness, 483
Kramer's theory of seasickness, 480
Kreidl's onychoscope, 482

Labyrinth, concussion of mechanisms of the, in boxing, 493
Labyrinthine receptors, varying sensitiveness of, 356
Lady Wolseley, steamship, observations on seasick subjects on, 417, 456–461
Literature of seasickness, 478–485
Localization theory of seasickness, the, 485
Lorna Doone, steamship, observations on seasick subjects on, 378
Lump-sensation in stomach in seasick patients, 340–401

Maggie, motor launch, observations on subjects for seasickness on board of, 343, 430, 431
Maillet's theory of seasickness, 485
Medical colleges should give special instruction for treating seasickness, 515
Medullary centres, prolonged constant irritation of, at sea, 355

Nelken's theory of seasickness, 479
Nervous and muscular systems, tension on, in seasickness, 504
Nervous centres, exhaustion of, characteristic of seasickness, 419
Nervous manifestations of seasickness, 340
Nervous system, the effects of seasickness upon the, 513
Neurotic type, some persons of the, immune from seasickness, 487
Neuro-vascular mechanism, inefficiency of, 355
  tardiness of, in certain conditions, 362
Nystagmus in seasickness, frequent absence of, 502

Occipital headache in seasickness, 349
Ocular functions, disturbances of, and vertigo, 490
Odours, influence of, upon sufferers from seasickness, 505
Old age frequently immune from seasickness, 508

Paræsthesiæ of the scalp, a symptom of seasickness, 349
Pellarin's theory of seasickness, 479
Perversion of sensory function, in seasickness, 340
Pfanz's theory of seasickness, 482
Phenomena, characteristic, of seasickness, 344, 345
Physicians, grave responsibility of, in sending patients to sea, 514
Pitching of ship as a secondary cause of seasickness, 499
Pliny on seasickness, 478
Pollard's theory of seasickness, 479
Protocols of observations on seasick subjects, 421–477
Psychic depression and disagreeable sights, 419
Psychic factor, the, in seasickness, 486, 487
Psychic theory of seasickness, 485

Psychic therapeutics, amusing incident in, 371
Pulse, concomitant slowing of, in seasickness, 341
Pulse-rate, variation of, in seasickness, 344
Pulse-rate and blood-pressure, variations of, in seasickness, 347
Pulmonary tuberculosis on board ship, case of, 516, 517

Quatre Frères, fishing smack, observations of seasick subjects on, 368, 370, 417, 465, 466

Recovery from seasickness, 419
Reflexes, lethargy of and "warming up" of athletes, 363
Regnault's theory of seasickness, 484
Roesen's theory of seasickness, 484
Rolling of a ship the least effective motion in producing seasickness, 499
Rosenbach's theory of seasickness, 480

Salicyluric acid in urine, 348
Salol motility tests, 342–409
Savory's theory of seasickness, 481
Schwerdt's theory of seasickness, 481
Sea, injudicious travelling on, 512
Seasick patients, effect of tobacco-smoke on, 340
Seasickness, a puzzle to mankind, 507
  aggravated by bad management, 521
  best time to study circulation in, 360
  can be effectively prevented by judicious measures, 518
Seasickness, circulatory and gastric disturbances in, 487
  common features of, 347
  conflicting reports as to immunity, 508
  developing a susceptibility for, 419
  digestive disturbances in, 340
  digestive symptoms of, 348
  during a transatlantic trip, 345–356
  effects of, and their relation to diseased conditions, 512–517
  effects of, upon pregnant women, 518
  effects of, upon the nervous system, 512
  effects of, upon the organism, 417
  etiology of, 486–507
    eye disturbances as a cause of, 491
  history and literature of, 478–485
  horrors of, 369
  hot weather, effect of, on, 488
  how recovery from, takes place, 518–525
  immunity of animals to, 510, 511
  important clue as to methods of treatment, 347
  influence of imagination in etiology of, 347
  nature's most effective way in circulatory depression of, 389
  nervous manifestations of, 340
  not due primarily to disturbance of circulation, 381, 417
  on the occurrence of, 508–511
  outlines of treatment of, 518–525
  phenomena of, 340, 344

Seasickness, predisposition to, 509
  prime cause of, 340
  prolonged exposure to conditions that cause, 417
  psychic factor in, 486
  recovery from, 419
  slowing of the pulse in, 341
  studies in, 339–421
  subconscious memories of past experiences of, 347
  subjective phenomena of, 360
  subjective symptoms of, 342
  symptoms of, 349
  the true cause of, 495
  theories of, 478–485
  treatment of an actual attack of, 521
  value of atropin in warding off disagreeable effects of, 376
  various devices employed to prevent, 520
  victims of, 419
Self-destruction the result of poor medical treatment in seasickness, 514
Semicircular canals affected in seasickness, 502
Sewanas' theory of seasickness, 479
Shelmerdine's theory of seasickness, 483
Ship at sea, motions of, 499
Ship, stops and starts in motions of, tend to bring on seasickness, 500
  upward and downward motions of, may affect otoliths, 501
Ship surgeons require special training, 515
Shock, condition of, in seasickness, 492
  the mechanism of, 389
Simon's theory of seasickness, 484
Skin, tonic hyperemia of, 355
Somatic theory of seasickness, 485
Southwestern, steamship, observations on seasick subjects on, 371, 373–375, 377, 378, 462, 463, 466, 467
Spiral movement, most effective for producing sickness, 499, 501
Stomach, a secondary source of irritation in seasickness, 521
Stomach tube should not be used in seasickness, 522
Subconscious memories of past experiences of seasickness, 347
Subjective phenomena of seasickness, 340, 351, 353
Suicide at sea, cause of, 513, 514
Sumner's theory of seasickness, 481
Susceptibility to seasickness, developing a, 419
Swinging beds, chairs, etc., to prevent seasickness, 520
Symptoms of seasickness, 342, 349

Taurus, steamship, observations on seasick subjects on, 339, 421
Teutonic, steamship, observations on seasick subjects on, 380, 467–477
Theories of seasickness, 478–485
Thoma's theory of seasickness, 484
Tiger, steamship, observations taken on seasick subjects on board of, 454–456
Tobacco-smoke, effect of, on seasick patients, 340, 345
Tousey's theory of seasickness, 480
Toxic acoustic neuritis, case of bilateral deafness caused by, 503

Transatlantic trip, studies of seasickness during a, 345–356
Travelling at sea, improvement of general conditions of, 514
Treatment of seasickness, important clue in, 347

Urine diminished during seasickness, 516
U-tubes, system of, to prevent seasickness, 524

Van Trostenburg's theory of seasickness, 485
Vaso-motor exhaustion, result of, 351
Vaso-motor mechanism, activity of, 340
  lethargy of, 362
Vaso-motor system, exhaustion and recovery of, 354
  influence of cold weather on the, 348
  interesting feature in the physiology of the, 362
Vertigo, causation of, 506
Vestibular terminals, degeneration of, 489
Vestibulo-cerebellar balanced mechanisms, disturbance of, 364
Vestibulo-cerebellar mechanisms, 497
Victims of seasickness may become immune, 419
Vomiting, nature's most effective way in relieving medullary anæmia, 389
Vomiting of the cerebellar type, 498

Waugh's theory of seasickness, 483
Weitlauer's theory of seasickness, 482
Well-marked nystagmus in seasickness, absence of, 502
Wollaston's theory of seasickness, 478
Women, smoking on deck offensive to, 524

Zingher's theory of seasickness, 484

INDEX TO AUTHORS REFERRED TO IN SEASICKNESS

Barker, 479, 516
Barnett, 484, 486
Bayliss, 384
Beard, 480, 508, 520
Berard, 479
Berillon, 484
Bickel, 502, 506
Bier, 484
Binz, 482, 483
Bonnet, 484

Castelet, D'Ailhaud, 480
Cato, 478
Chapman, 479
Clark, 499
Cornelius, 482
Corning, 483, 484
Crile, 389

Darnall, 481
Darwin, Erasmus, 478
De Cyon, 490
De Vries, 481

Edkins, 384, 393
Ewald, 487, 490

Farez, 484
Ferrier, 498
Flasschoen, 484
Flourens, 491, 507
Frahm, 524

Guien, 479
Guillabert, 479

Hagen-Torn, 483
Hazen, 481
Helmholtz, 507
Henderson, 389
Hippocrates, 478
Högyes, 487

James, 487
Jendrassik, 404
Jobert, 479

Klein, 481
Koch, 495
Koenig, 519
Koepke, 483
Kramer, 480
Kreidl, 487

Luciani, 504

Maillet, 485

Nelken, 479

Pellarin, 479
Pfanz, 482, 483
Pliny, 478
Pollard, 479
Porter, 389

Regnault, 484
Roesen, 484
Rosenbach, 480, 485, 495

Saar, 522
Savory, 481, 482, 486
Schiff, 487
Schwerdt, 481
Sewall, 498
Sewanas, 479
Shelmerdine, 483
Simon, 484
Starling, 384
Stevens, 490

Sumner, 481

Thoma, 484
Tousey, 480

Unger, 522

Van Renterghem, 484
Van Trostenburg, 485

Waugh, 483
Weir-Mitchell, 504
Weitlauer, 482
Witmaack, 489, 503, 509
Wollaston, 478

Zingher, 484
Zuelzer, 522

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